

**CLINICAL AND HISTOPATHOLOGICAL PROFILE IN
PATIENTS WHO UNDERWENT RENAL BIOPSY IN A
TERTIARY CARE CENTRE**

**Dissertation submitted to
THE TAMILNADU DR. M.G.R MEDICAL UNIVERSITY
CHENNAI**

**In partial fulfillment of regulations
For award of the degree of
M.D (GENERAL MEDICINE)**



**KILPAUK MEDICAL COLLEGE
CHENNAI-600 010**

April 2016

BONAFIDE CERTIFICATE

This is to certify that dissertation named “**CLINICAL AND HISTOPATHOLOGICAL PROFILE IN PATIENTS WHO UNDERWENT RENAL BIOPSY IN A TERTIARY CARE CENTRE**” is a bonafide work performed by DR.S.SETTU, post graduate student, Department of Internal Medicine, Kilpauk Medical College, Chennai-10, under my guidance and supervision, in fulfilment of regulations of the Tamil Nadu Dr. M.G.R Medical University for the award of M.D. Degree Branch I (General Medicine) during the academic period from July 2013 to June 2016.

Prof.Dr.R, VENKATRAMAN, M.D., D.M

Professor and HOD,
Dept of Nephrology,
Government Royapettah Hospital.
Kilpauk Medical College.

Prof.Dr.S.MAYILVAHANAN, M.D

Professor of Medicine,
Government Royapettah Hospital,
Kilpauk Medical College

Prof.Dr.S.USHALAKSHMI, M.D, FMMC

Professor and Head of the Department,
Department of Medicine,
Kilpauk Medical College.

Prof .Dr.R.NARAYANABABU, M.D., DCH.

THE DEAN,
KILPAUK MEDICAL COLLEGE,
CHENNAI-10

DECLARATION

I solemnly declare that this dissertation named **“CLINICAL AND HISTOPATHOLOGICAL PROFILE IN PATIENTS WHO UNDERWENT RENAL BIOPSY IN A TERTIARY CARE CENTRE”** was prepared by me at Government Kilpauk Medical College and Hospital, Chennai, under the guidance and supervision of Prof.Dr.R.VENKATRAMAN,M.D.,D.M, professor/HOD, Dept. of Nephrology and Prof.Dr.S.MAYILVAHANAN,M.D., Professor, Department of Internal Medicine, Government Royapettah Hospital, Chennai.

This dissertation is submitted to **The Tamil Nadu Dr.M.G.R.Medical University, Chennai** in partial fulfilment of the University regulations for the award of the degree of **M.D. Branch I (General Medicine)**.

Place: Chennai

Date:

(Dr.S.SETTU)

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ABBREVIATIONS

SD - STANDARD DEVIATION

CAD - CORONARY ARTERY DISEASE

SLE - SYSTEMIC LUPUS ERYTHEMATOSIS

HE - HEMATOXYLLINE AND EOSINE

PAS - PERIODIC ACID SCHIFF

GBM -GLOMERULAR BASEMENT MEMBRANE

MCD -MINIMAL CHANGE DISEASE

FSGS -FOCAL SEGMENTAL GLOMERULOSCLEROSIS

MN -MEMBRANOUS NEPHROPATHY

MPGN- MEMBRANO PROLIFERATIVE GLOMERULONEPHRITIS

PIGN -POST INFECTIOUS GLOMERULONEPHRITIS

LN -LUPUS NEPHRITIS

LM- LIGHT MICROSCOPE

EM- ELECTRON MICROSCOPE

HIV - HUMAN IMMUNODEFICIENCY VIRUS

NSAID- NON STEROIDAL ANTI INFLAMMATORY DRUGS

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- ii. QUESTIONNAIRE PROFORMA**
- iii. ETHICAL COMMITTEE**
- iv. PLAGIARISM CERTIFICATE**

INTRODUCTION

The kidney is the mysterious organ that has a major role in excreting waste products, regulating body fluids and balancing soluble ions in the body⁽¹⁾. The object of this study is to assess clinical and histopathological profile of kidney disease in patients who underwent renal biopsy and differential diagnosis of the common renal disease and interpretation of these results.

The beginning of systematic study of Proteinuria and haematuria can be dated back to Sir Richard Bright who is referred to as the Father of Nephrology⁽²⁾. The persistently increased 24 hr. protein excretion (150mg/day) OR the urine protein/creatinine ratio in a random urine sample of greater than 0.2g is usually a marker of the kidney damage. Increased excretion of albumin (>30mg/day) is a sensitive marker of chronic kidney disease due to diabetes, glomerular disease and hypertension. Also increased excretion of low molecular weight globulins is a sensitive marker for few types of tubulo-interstitial diseases.

Haematuria can be microscopic or macroscopic and it can occur in various glomerular and tubulointerstitial diseases of the kidney. Though haematuria occurs mostly in nephritic syndrome, sometimes it may also occur in nephrotic syndrome. Renal biopsy can be done in patients with some systemic disorders where knowing the nature of renal involvement has a great

therapeutic and prognostic implication. It can also be done in the patients who fail to improve in spite of optimum therapy. So correlating Proteinuria, haematuria, oliguria, oedema and other comorbid conditions with the pathology found on renal biopsy will help us in a better understanding of the kidney diseases. Thus one can take preventive as well as early therapeutic measures to delay the consequences of the kidney diseases.

AIM OF THE STUDY

To study the clinical and histopathological profile of the patients who underwent renal biopsies.

REVIEW
OF
LITERATURE

OVERVIEW

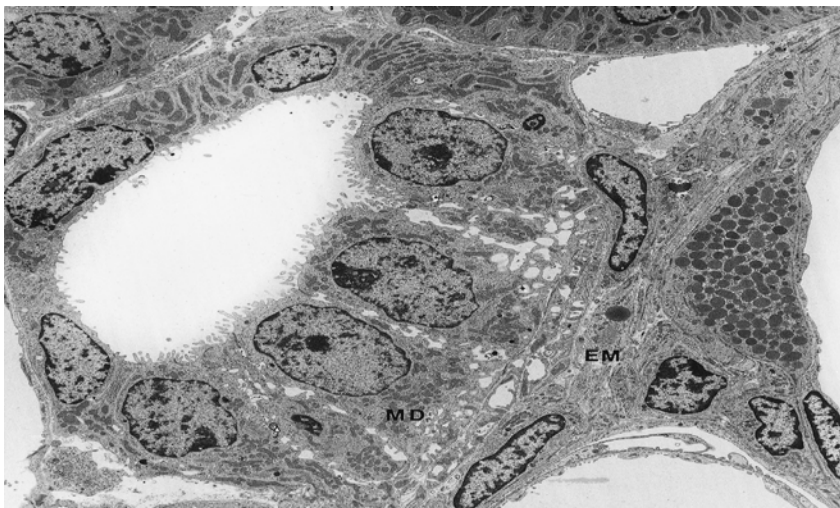
1. Nephrons

The functional and the basic unit of the kidney is the nephrons⁽³⁾ which are composed of renal corpuscle (glomerulus and Bowman's capsule), and the cylindrical epithelial-lined tubular component (proximal tubule, Henle's loop and the distal tubule).

2. Kidney histology

2.1 Glomerulus

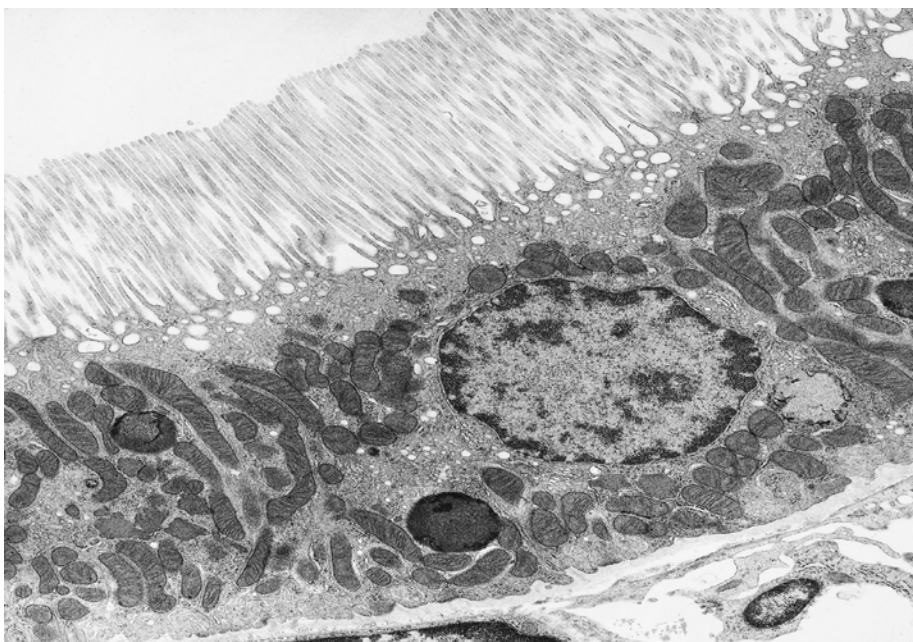
Glomerulus was first described by Malpighi⁽⁴⁾ and then it was clearly explained by Bowman^(5,6). It contains glomerular tuft and Bowman's capsule which is the dilated part of proximal tubule⁽⁷⁾. Glomerulus is lined internally by endothelium and it is supported by mesangium, the matrix and number of cells.



Electron micrographic appearance of the juxtaglomerular apparatus

2.2 Proximal tubule

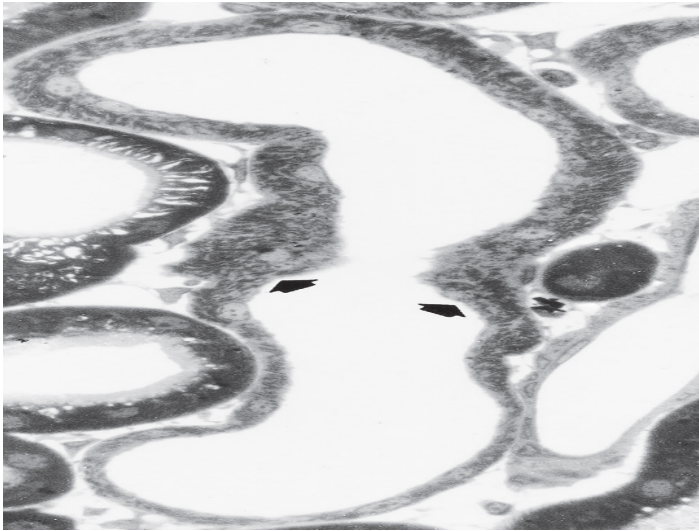
It contains two parts. One is convoluted part and another one is straight part⁽⁸⁾. The convoluted part is situated in cortex and the straight part is situated in medulla. Its total length is 11micrometer. It's lined by columnar or cuboidal cells. The major function of proximal tubule is reabsorption of almost more than 60% of glomerular ultra-filtrate.



Electron micrographic appearance of the proximal tubule

2.3 Henle`s loop

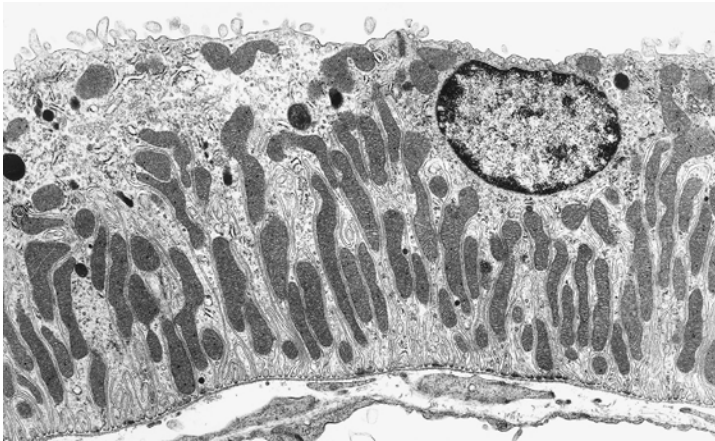
Henle`s loop is situated between the proximal tubule and the distal tubule. It is a U shaped loop. It is lined by flat type cells without brush border. They transport the water, sodium and chloride passively.



Thick ascending limb of loop of henle

2.4 Distal tubule

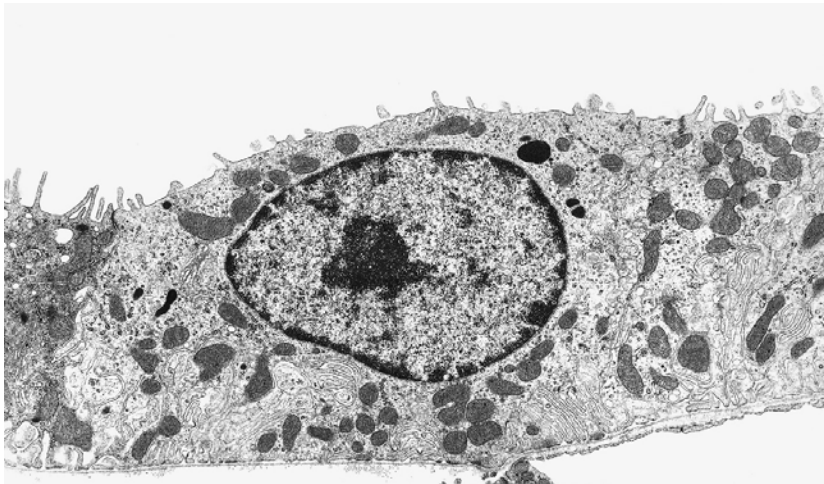
Distal tubule is lined by cuboidal cells with eosinophil rich cytoplasm. It involved in active transport of sodium chloride, which is the main function of distal tubule⁽⁹⁾. It is connected distally to collecting ducts.



Pars convoluta segment of the distal tubule.

2.5 Collecting duct

Collecting duct has three parts, 1) outer medullary part, 2) inner medullary part, 3) cortical part⁽¹⁰⁾. First two parts are lined by different epithelial cells. Cortical part is lined by cuboidal cells without brush border. Two types of cells are present in collecting ducts the principal cells and the intercalated cells. P cells secrete the potassium into the cortical collecting duct. I cells have many enzymes like carbonic anhydrase II⁽¹¹⁾.



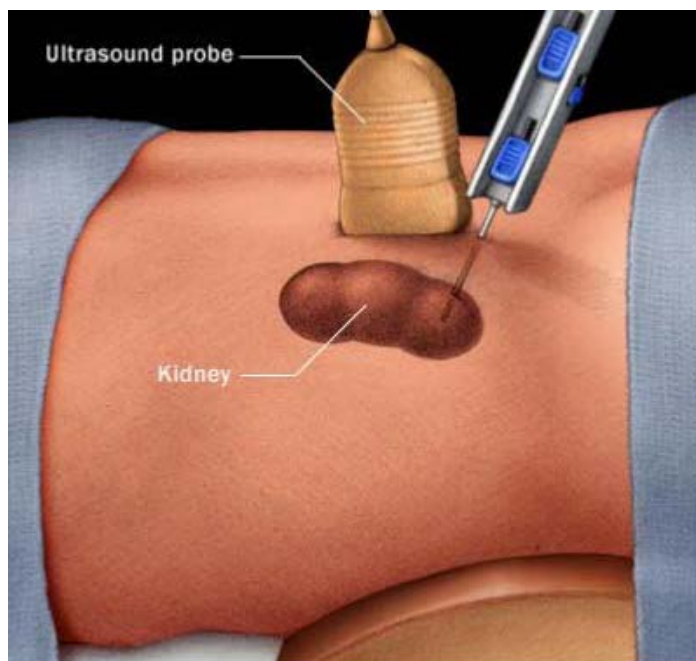
Principal cell from cortical collecting duct

5. Renal biopsy

First renal biopsy was done in United States a hundred years before⁽¹²⁾. But its clinical use became more after 1950s^(13, 14). It is useful to localise the lesion, to quantify and qualify the lesion, to know the extent of the lesion and also to know the response to the treatment and to follow up the patient.

7. Biopsy technique

It can be done either by Nephrologist or Radiologist. In olden days it was done by true cut method. But nowadays it is done by biopsy gun⁽¹⁵⁾. It is done in prone position for native kidney and in supine position for transplanted kidney. It is done under local anaesthesia with ultrasound guidance by using 16 or 14 gauge needle⁽¹⁶⁾. But for biopsy of renal mass 18-21 gauge needles are better choices^(17,18,19). The appropriate place of taking biopsy is juxtamedullary, because glomeruli will involve earlier in focal segmental glomerulosclerosis.



Biopsy technique

8. Risks

Renal biopsy is a painful procedure. The most common complication is microscopic haematuria^(20,21). Other complications^(22,23,24,25) are macroscopic haematuria, perinephric hematoma, arteriovenous fistula, renal pelvic damage, injury of adjacent organs, need of blood transfusion, renal tissue loss and rarely death. These complications can be minimised by ultrasonogram guidance.

9. Gross inspection

After taking biopsy, the biopsy material should be placed in a drop of normal saline and it should be examined under microscope for colour and appearance. While handling the specimen one should not use forceps, because it will cause artefacts^(26,27,28). Red coloured hemispherical tissues are glomeruli. The biopsy material should be divided fixed in a proper solution without any delay, to prevent drying of the specimen. The various stains used for staining are HE stains, Methenamin silver, Masson Trichrome, periodic acid Schiff, Congo red and Reticulin.

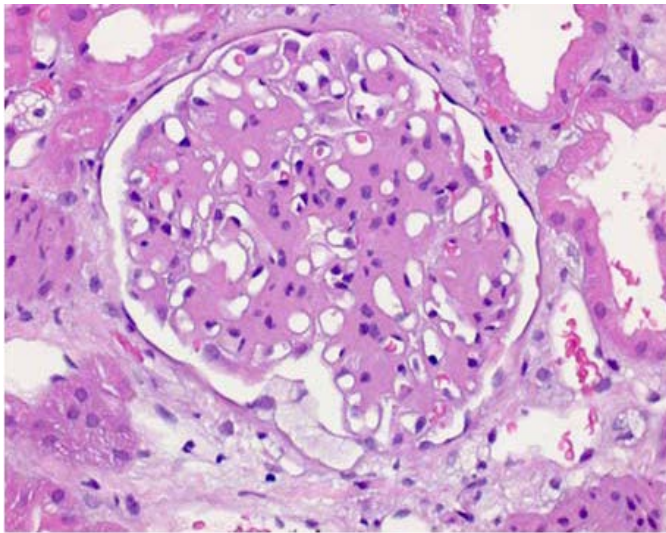
10. Fixatives

Neutral buffered formaldehyde is suitable for light microscope, immunohistochemistry and molecular studies. Mercury based fixatives like Zenker's, Karnowsky's and Bouin's fluid are appropriate for light microscope. But they are not preferred fixatives for immunohistochemistry and molecular studies. A modified Carnoy fixative is used for both light microscope and electron microscope and it is called as Methacaren^(27,28). Microwave devices are used for processing and fixing, whenever immediate results are required. 2-3 % glutaraldehyde is usually used for electron microscope. Immunofluorescence samples should not be fixed. They should be sent in Michel's media.

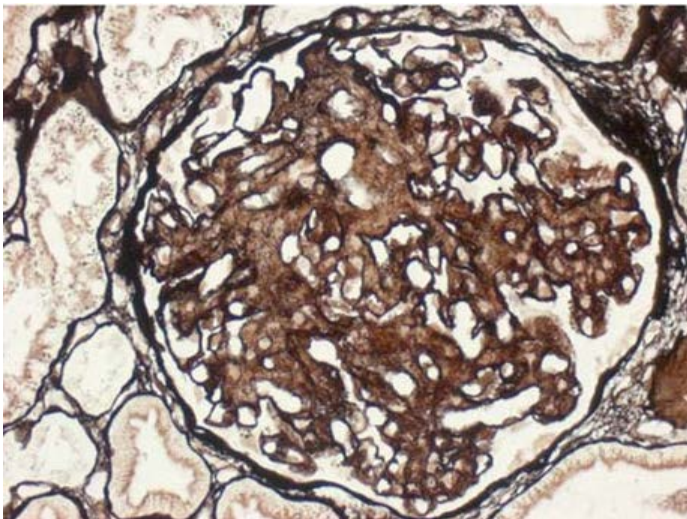
11. Sectioning and staining

After the histological processing and paraffin embedding, the tissues are sectioned by the microtome. Then these sections are prepared as thin as 3 μm or less for the light microscopy. Thicker sections are needed for Congo red and for immunohistochemistry staining. The most useful stains for the light microscopy are Haematoxylin and Eosin (H&E), Periodic Acid Schiff (PAS), Trichrome, Congo red, Methanamine silver and Reticulin. H&E highlights mainly the cells well, while Methenamine silver reveals mainly the basement membrane and the matrix of connective tissues. In PAS staining, there will be good highlighting

of cells and the basement membrane. Trichrome staining is suitable for the basement membrane, fibrosis and for the assessment of deposits. Congo red is used for amyloid discovering while elastin stains like Reticulin are helpful for identifying vascular lesions



HE staining



Silver staining

12. Specimen adequacy

There is always a question of how much tissue is necessary for the diagnosis of renal disease. In the diffuse glomerular diseases, such as Amyloidosis and the membranous glomerulopathy, one glomerulus is enough for the diagnosis. In the diagnosis of focal diseases, considering the random distribution of the abnormalities, the probability of finding any glomerulopathy is represented by the binomial equation ^(29,30,31). For example, if we have 10 glomeruli and disease exist in 10% and 35% of them, we will be having 65% and 95% positive report in the biopsy respectively.

Fogo mentioned 25 and 10 glomeruli are needed for the most accurate diagnosis in light microscopy for the native and the transplanted kidneys respectively. In a study it was noted that a specimen with at least 25 glomeruli are needed for the diagnosis of chronic lesions of the kidney ^(32,33). There is some semi-quantification of pathologic findings including glomeruli number, mesangial matrix volume, inflammatory cell infiltrate, percentage of fibrosis, percentage of affected glomeruli and atrophy in different patients. Also some quantification techniques in renal biopsy have been reported ^(32,33,34,35). These quantifications techniques are helpful in monitoring the patients and their response to the therapy as well as comparison of the different biopsies samples and their correlation with the clinical points. The quantification methods require a standard protocol for the processing and the sectioning.

13. Differential diagnosis of various renal lesions

Renal biopsies are not only useful in diagnosis and differentiating various diagnosis but also useful in knowing the severity and extent of the disease and to quantify the irreversible scarring. There are four main parts in the kidney which include glomeruli, interstitium, tubules and vascular part. Primary changes will occur in any of these structures and secondary changes will occurs in other structures. So meticulous and stepwise approach is needed to identify the primary and secondary changes. Glomerular changes are inflammation, basement membrane changes, scarring, spikes, fibrinoid exudates,deposits and hypercellularity.Tubular changes are cellular injuries, atrophy, regeneration, cast, edema,fibrosis and crystals.Interstitium pathologiesinclude cellular infiltrate, edema, and fibrosis^(36,37,38). Vascular partchanges are inflammation, sclerosis, thrombosis and hyalinosis.

14. Glomerular lesions

There is a standard terminology for the glomerular involving lesions report of which is stated by Jennette and et al^(39,40,41).

- **Focal**- less than 50% of glomeruli involvement
- **Diffuse**-more than 50% of glomeruli involvement
- **Segmental**- part of a glomerulus involvement
- **Global**- all of the glomerulus are involved
- **Mesangial hypercellularity**-four or more nuclei in the mesangial region

- **Endocapillary hypercellularity:** means increased cellularity internal to the GBM composed by leukocytes, endothelial cells or mesangial cells.
- **Extracapillary hypercellularity:** means increased cellularity in Bowman's space (more than the onelayer of the parietal or the visceral epithelial cells or monocyte/macrophage).
- **Crescent:** means extracapillary hypercellularity other than the epithelial hyperplasia of the collapsing variants of FSGS.
- **Fibrinoid necrosis:** means lytic destruction of cells and matrix with deposition of the acidophilic fibrin-rich material.
- **Sclerosis:** means increased collagenous extracellular matrix that is expanding to the mesangium, obliterating capillary lumens or forming adhesions to the Bowman's capsule
- **Hyaline:** means glassy acidophilic extracellular material.
- **Membranoproliferative:** means combined capillary wall thickening and the mesangial or endocapillary hypercellularity.
- **Lobular (hypersegmented):** means expansion of segments that are demarcated by intervening urinary space.
- **Mesangiolysis**^(42,43,44,45,46,47)-detachment of paramesangial GBM from the mesangial matrix or lysis of the mesangial matrix.
- **Focal glomerulonephritis-** includes inflammatory lesions in less than 50% of the glomeruli.

The differential diagnoses are categorised based on the age and are as follows

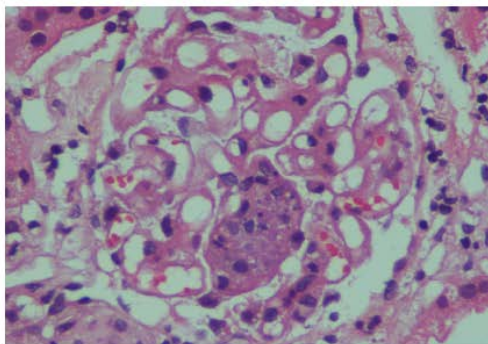
<15 years

1. IgA nephropathy Immunofluorescence micrograph of IgA nephropathy

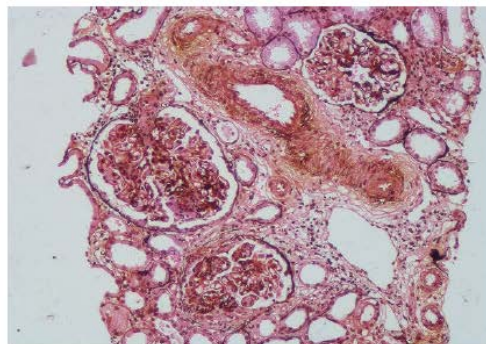
15-40 years

1. thin basement membrane disease
2. IgA nephropathy
3. systemic lupus erythematosus

HE



PASM



49 years female with diffuse proliferative lupus nephritis -IV

4. mesangial proliferative glomerulonephritis
5. hereditary nephritis

>40 years

1. IgA nephropathy

Diffuse glomerulonephritis -will affect most or all of the glomeruli and the differential diagnoses based on the age are:

<15 years

1. membranoproliferative glomerulonephritis
2. post infectious glomerulonephritis

15-40 years

1. Post infectious glomerulonephritis
2. Systemic lupus erythematosus

>40 years

1. Post infectious glomerulonephritis
2. Rapid progressive glomerulonephritis
3. vasculitis
4. Fibrillary glomerulonephritis

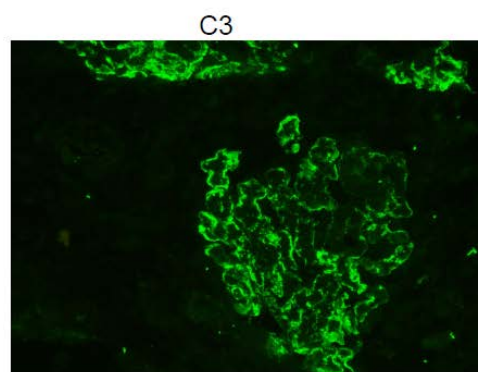
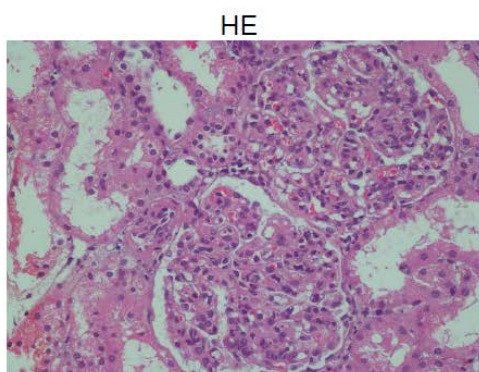
Nephrotic syndrome

Associated with proteinuria, lipiduria and hypertension

Differential diagnoses according to the age include

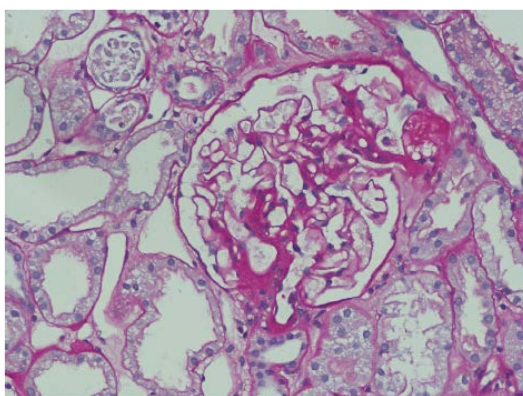
<15 years

1. Minimal change disease
2. Mesangioproliferative glomerulonephritis



35 year female with hypothyroidism, hypertension and hematuria

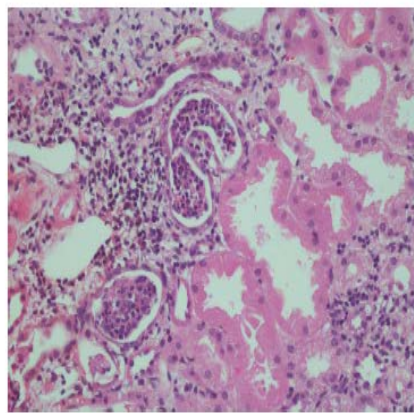
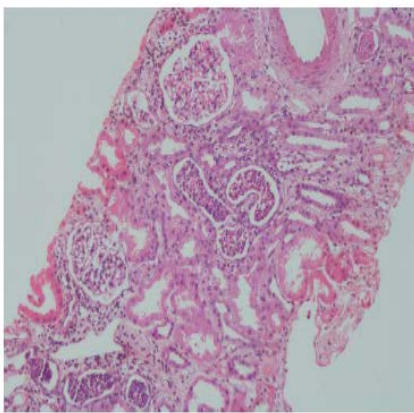
3. Focal segmental glomerulosclerosis



40 year old male diabetic with proteinuria-FSGS

15-40 years

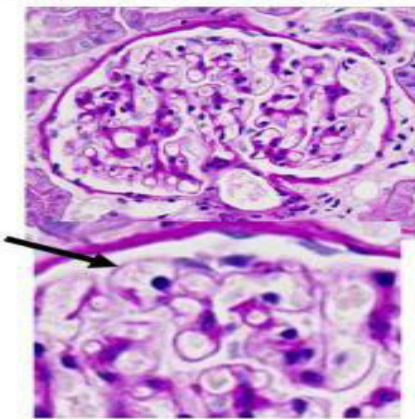
1. Diabetic nephropathy
2. Preeclampsia
3. Post infectious glomerulonephritis
4. Focal segmental glomerulosclerosis
5. Minimal change disease



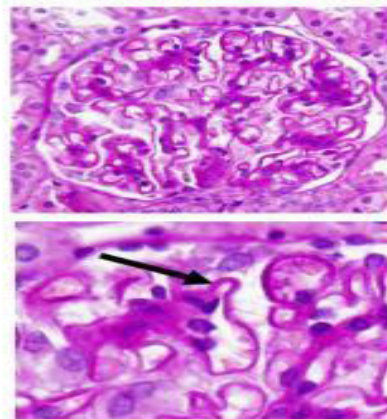
26 year female with anasarca-MCD

6. Membranous nephropathy

Normal glomerular capillaries with thin walls

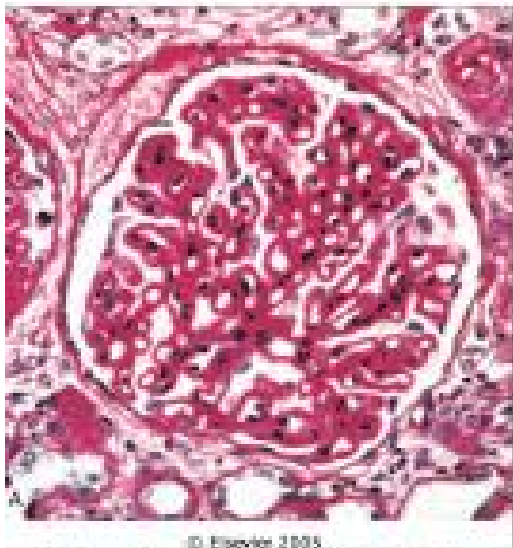


Membranous glomerulopathy with thick capillary walls

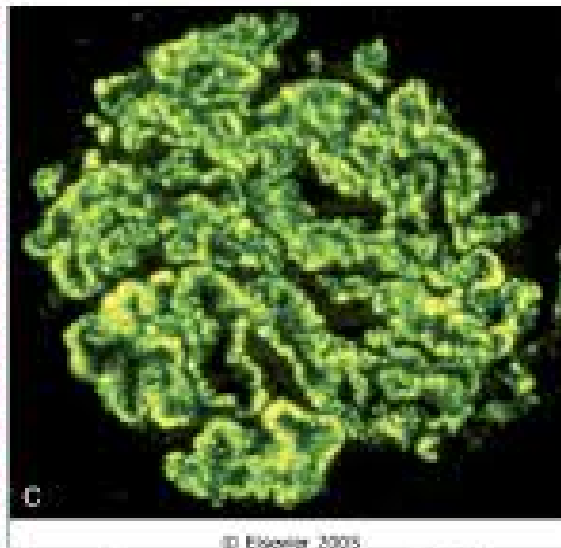


>40 years

1. IgA nephropathy
2. Focal segmental glomerulosclerosis
3. Amyloidosis
4. Post infectious glomerulonephritis
5. Membranous nephropathy



Light Microscopy (PAS Stain)



Immunofluorescence (IgG)

The glomerulus lesions are categorized -

Sclerosis

1. Collapsing
2. Usual
3. Secondary
4. Tip lesion of FSGS

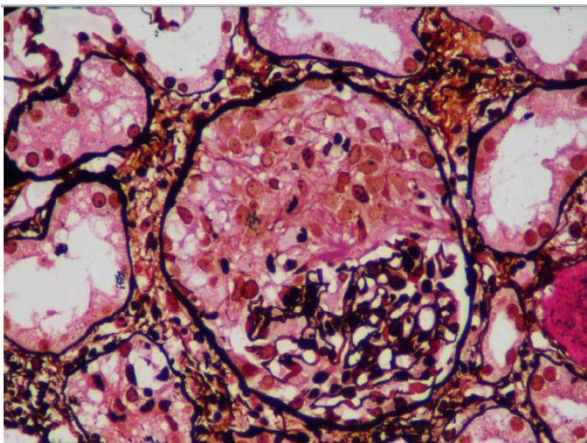
Crescent

According to cellular and degree of fibrous:

1. Cellular
2. Fibrous
3. Fibrocellular

According to immune deposits

1. Pauci immune



40 years old female presented with hematuria-pauci immune crescentic glomerulonephritis

Proliferation

Mesangial with nodules

1. Membranoproliferative glomerulonephritis
2. Chronic infection related glomerulonephritis
3. IgA nephropathy

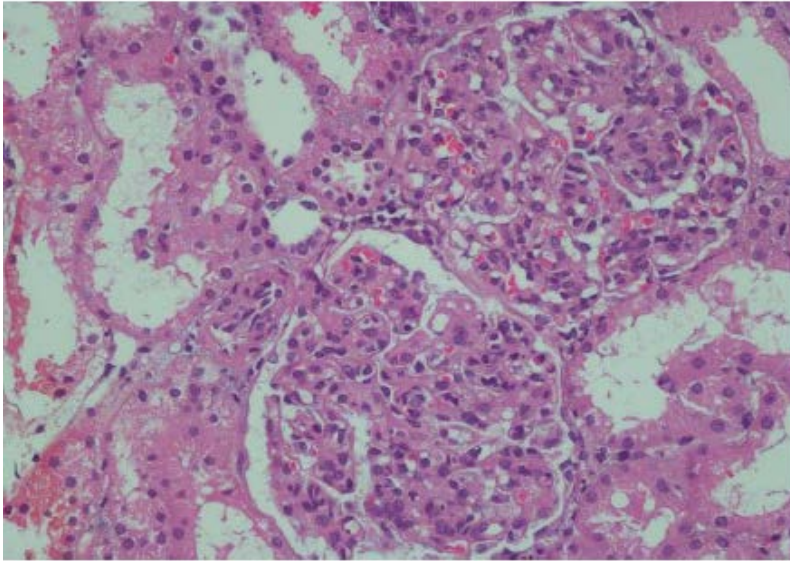
Mesangial without deposits

1. Focal segmental glomerulosclerosis
2. Minimal change disease
3. Early diabetic nephropathy

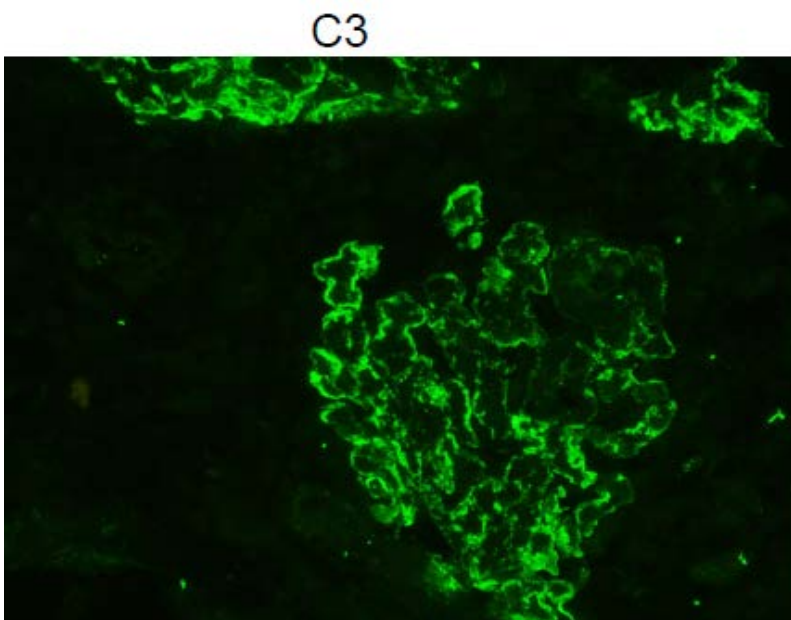
Mesangial and endocapillary

1. Proliferative lupus nephritis
2. Membranoproliferative glomerulonephritis
3. Dense deposit disease
4. Cryoglobulinemia glomerulonephritis
5. immunotactoid glomerulopathy

6. Postinfectious glomerulonephritis



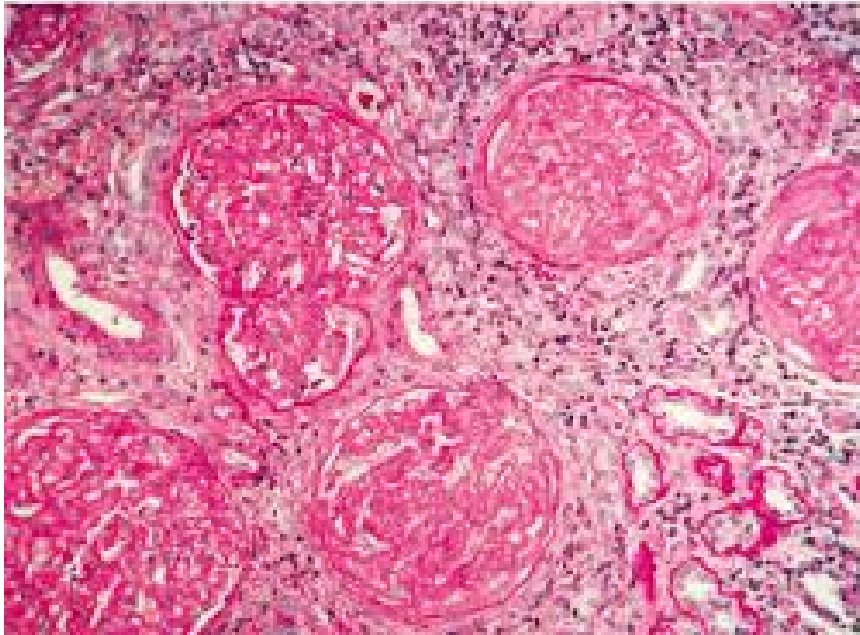
35 years female - LM of PSGN showing influx of neutrophils



IF microscopy of the same patient showing C3 deposits

7. fibrillary glomerulonephritis

E



LM picture of fibrillary glomerulonephritis

Unusual lesions

1. Foamy macrophages intraglomerular:
2. Foamy podocytes

It might be remembered that each specific histological pattern in the light microscopy could be seen in different diseases

No abnormality

1. minimal change disease
2. Thin basement membrane nephropathy

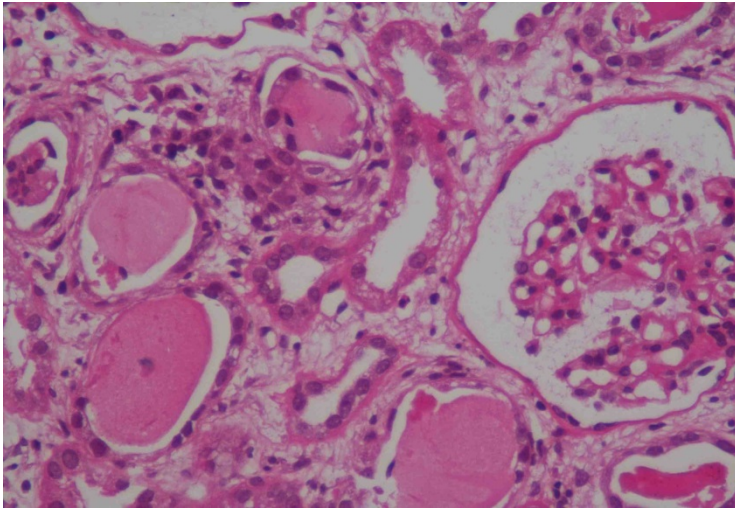
Thick capillary walls only

1. Thrombotic microangiopathy
2. Membranous glomerulopathy

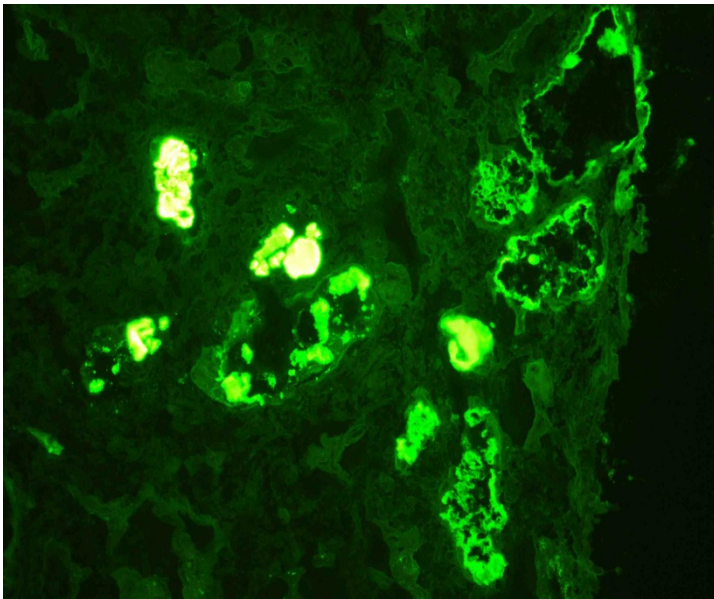
Thickened walls with mesangial expansion without the hypercellularity

1. Diabetic glomerulopathy
2. Membranous glomerulopathy with mesangial deposits
3. Amyloidosis
4. Fibrillary glomerulonephritis
5. Dense deposit disease

6. Monoclonal Ig deposition disease



LM picture of 47 year male with cast nephropathy due to multiple myeloma



IF picture of same patient showing lambda chain deposits

Focal segmental glomerulosclerosis

1. Healing of previous glomerular injuries
2. Minimal change disease
3. Hypertension
4. Hereditary nephritis
5. Chronic phase of the focal glomerulonephritis

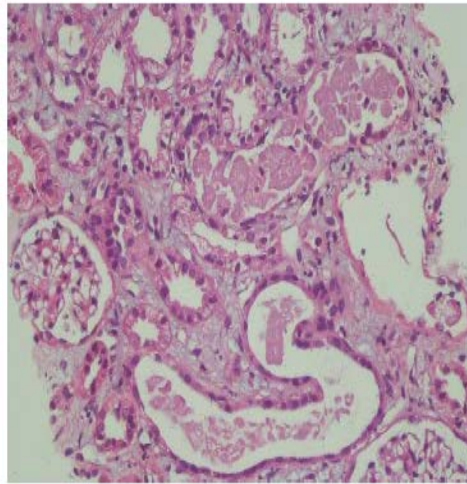
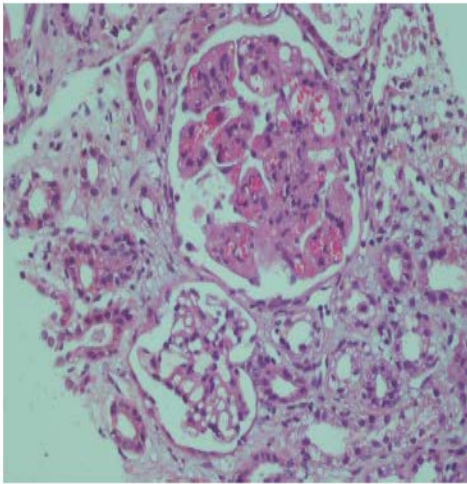
Membranous injury

1. Underlying malignancy
2. Drug consumption such as gold, penicillamine, mercury
3. Chronic hepatitis B
4. Systemic lupus erythematosus

Membranoproliferative glomerulopathy

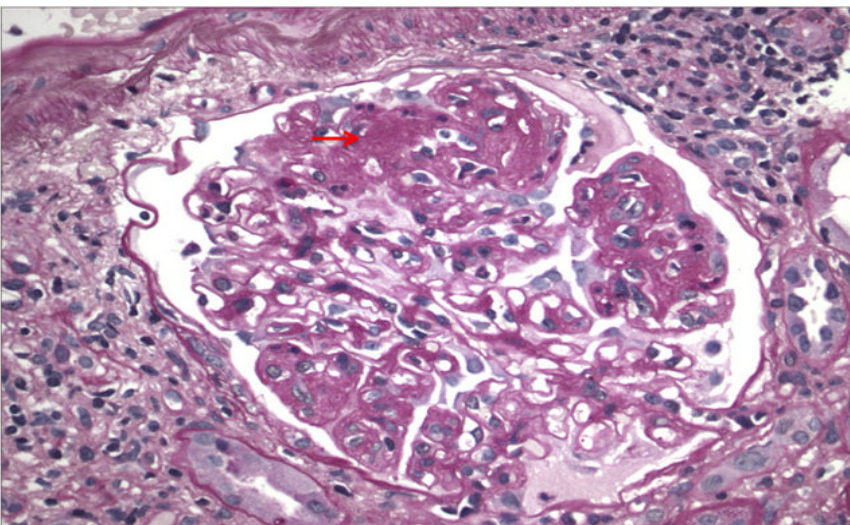
1. Membranoproliferative glomerulonephritis
2. Diabetic glomerulonephritis
3. Systemic immune complex disease
4. HepatitisC
5. Systemic lupus erythematosus
6. Infectious endocarditis
7. Fibrillary glomerulonephritis

8. Thrombotic microangiopathy



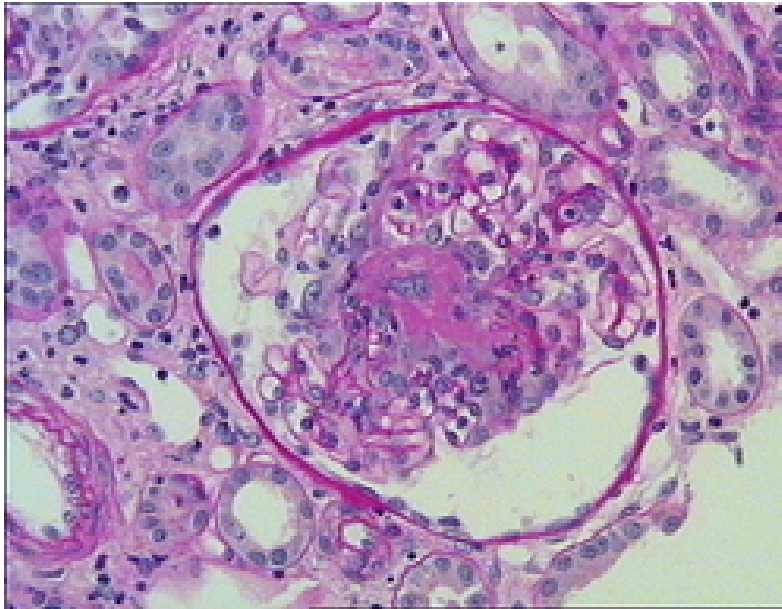
23years female with post LSCS AKI – fragmented RBC and thrombi

9. Immunotactoid glomerulopathy



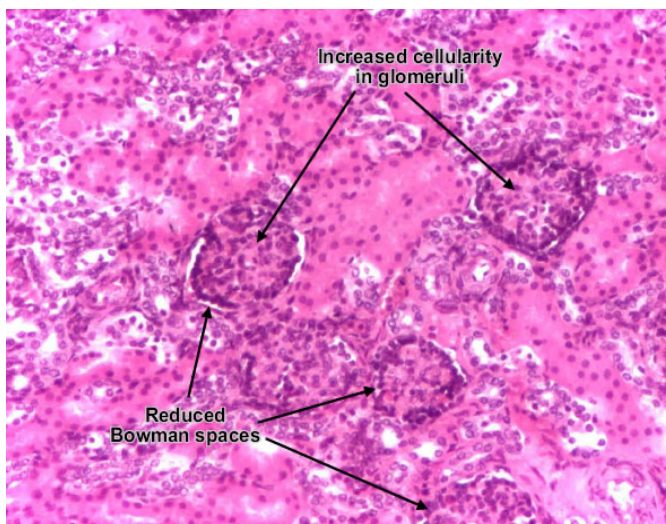
According to serologic studies

1. Anti GBM antibody: anti GBM antibody disease



LM picture of Anti GBM Antibody disease – shrinkage of glomerular tuft

2. Ant streptococcal antibody-post streptococcal glomerulonephritis



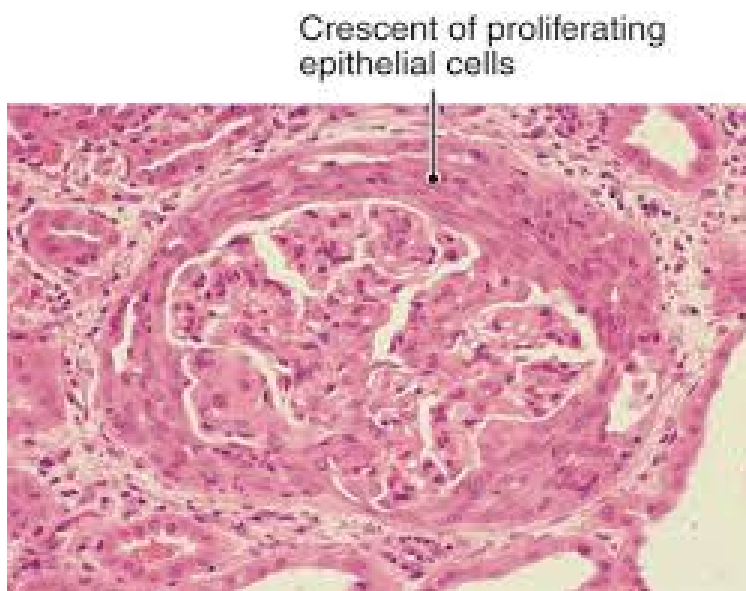
LM picture of PIGN

According to the decreased serum complement level

1. Mixed cryoglobulinemia
2. Postinfectious glomerulonephritis
3. Lupus nephritis
4. Membranoproliferative glomerulonephritis

According to the presence of acute renal failure

1. Minimal change disease with acute interstitial nephritis of NSAIDs
2. Idiopathic minimal change disease
3. Collapsing focal segmental glomerulosclerosis
4. crescentic glomerulonephritis



LM picture of crescentic glomerulonephritis

15. TUBULAR LESIONS

I. Acute tubular cell injury

1. Acute tubular necrosis
2. Hyaline droplet formation
3. Vacuolar change (fine and the diffuse appearance)
 - a. Fatty change
 - b. Foam cells
 - c. Hypokalemic nephropathy
 - d. Hydropic change

II. Tubular casts

Are principal histological feature of the light chain disease, myoglobinuria, hemoglobinuria, oxalate nephropathy, urate nephropathy, nephrocalcinosis and the drug-induced tubular lesions.

- Hyaline: seen in renal failure or low urine output states
- WBC: seen in tubulointerstitial inflammation
- Epithelial cell or granular: seen in acute tubular injury
- RBC: seen in glomerular bleeding
- Large hyaline fractured: seen in light chain casts (often accompanied by the giant cells and the neutrophils)
- Coarse granular acidophilic or red brick: indicates myoglobin or hemoglobin

III. Tubular atrophy

IV. Tubulitis

V. Tubular basement membrane changes

16.INTERSTITIUM LESIONS

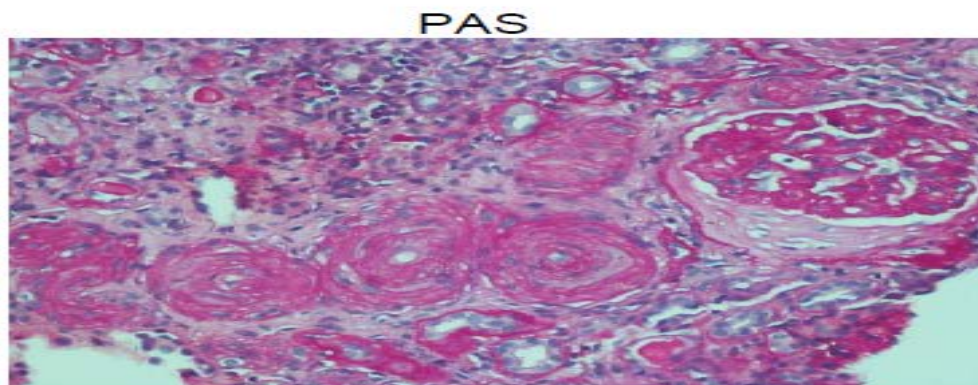
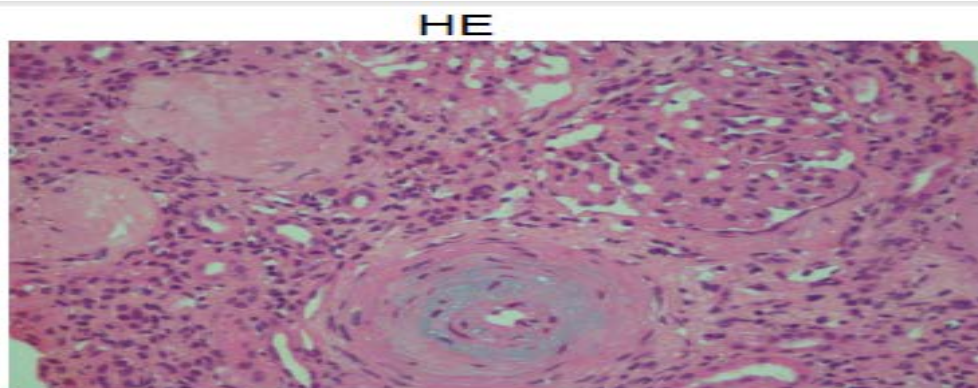
The lesions of the interstitium that can be observed in the light microscopy are listed here (5 types with modification).

- No pathologic changes
- Expansion and edema (due to the increased permeability of the vessels)
- Expansion with the leukocyte infiltration
- Foam cells
- Expansion with the neoplastic cells
- Crystals
- Fibrosis

17. VASCULAR LESIONS

The main injuries of the vascular elements are listed here (5 types with modification).

1. Vasculitis
2. Emboli
3. Deposition of the materials
4. Hypertension induced injuries



40 years male with hypertension – fibrinoid necrosis

METHODS AND MATERIALS

METHODS AND MATERIALS

This study is done on patients who underwent renal biopsy at the Department of Medicine and Nephrology, Government Royapettah hospital (GRH), Chennai. Number of patients studied was 50. They were in the age group 14-60 yrs. Out of 50 patients, 21 were female and 29 were male. The patients selected were presented with either nephrotic or nephritic features, with hematuria of either microscopic or macroscopic, Proteinuria of Nephrotic or nephritic range and renal failure. Biopsy done in post-transplant patients is excluded from the study.

Study group: patients underwent renal biopsy in government Royapettah hospital during the period from 2011 to 2014.

Study design: cross sectional study.

Study period: April-2015 to sep-2015

Place of the study: Government Royapettah hospital.

Conflict of interest: nil.

Hazards of the study: nil.

Sample size: 50

METHODOLOGY

After obtaining proper permission from Superintendent of the Government Royapettah hospital, details of the patients who underwent renal biopsy during the year from 2011 to 2014 were collected from medical records department in Government Royapettah hospital, which includes detailed presenting complaints, past history of hypertension, diabetes, dyslipidemia coronary artery disease, hypothyroidism, systemic lupus erythematosus, Amyloidosis, renal diseases, NSAID, use of native treatment, detailed personal history of diet pattern, alcohol abuse, smoking, illicit drug abuse, HIV, HEPATITIS viral markers status, examination findings like pallor, anasarca, skin lesions, blood pressure, laboratory investigations like urine routine, complete hemogram, random blood sugar, blood urea, serum creatinine, serum electrolytes, lipid profile, viral markers study for retrovirus and hepatitis viruses, ultrasonogram abdomen and special investigations like anti-nuclear antibodies in some occasions.

INCLUSION CRITERIA

Renal biopsy in Patients presented with nephrotic or nephritic features with microscopic or macroscopic hematuria and renal failure.

EXCLUSION CRITERIA

Renal biopsy in post-transplant patients

DATA COLLECTION

The data of the each patient was collected on a proforma specially designed for this study and the proforma contains demographic details of the patient, clinical details including history and examination particulars, laboratory investigation results and histopathological report of each patient.

STATISTICAL ANALYSIS

All the observations in this study analysed statistically using SPSS 20.0 software and CHI SQUARE test is used . P value of <0.05 is taken as significant.

OBSERVATION AND RESULTS

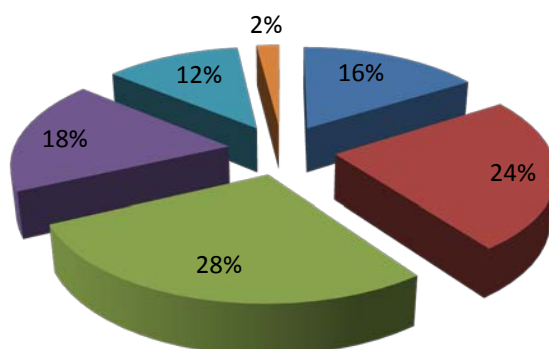
OBSERVATION AND RESULTS

AGE IN YEARS

AGE IN YEARS	FREQUENCY	PERCENT
Below 20	8	16.0
21-30	12	24.0
31-40	14	28.0
41-50	9	18.0
51-60	6	12.0
Above 60	1	2.0

AGE DISTRIBUTION

Below 20 21-30 31-40 41-50 51-60 Above 60

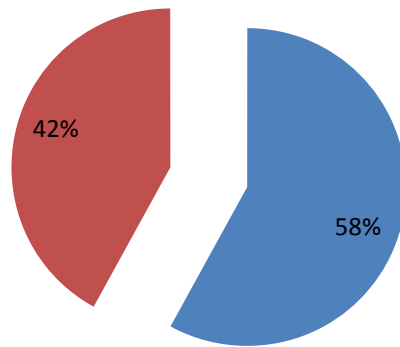


SEX

SEX	FREQUENCY	PERCENT
Male	29	58.0
Female	21	42.0

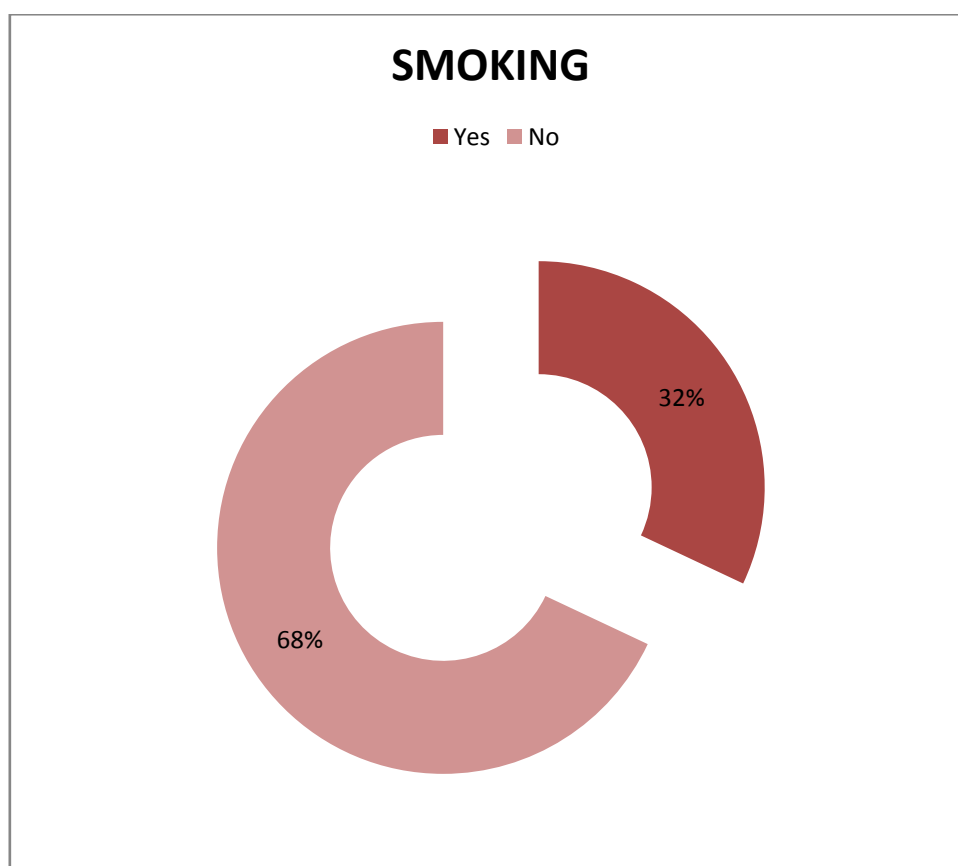
GENDER

■ Male ■ Female



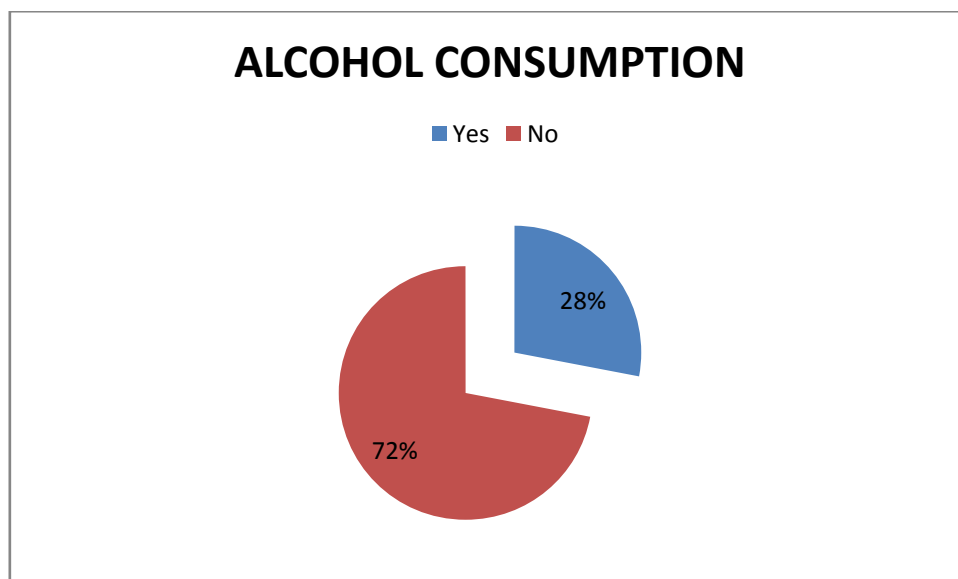
SMOKING

SMOKING	FREQUENCY	PERCENT
Yes	16	32.0
No	34	68.0



ALCOHOL

ALCOHOL	FREQUENCY	PERCENT
Yes	14	28.0
No	36	72.0



EDEMA

EDEMA	FREQUENCY	PERCENT
Yes	22	44.0
No	28	56.0

OLIGURIA

OLIGURIA	FREQUENCY	PERCENT
Yes	14	28.0
No	36	72.0

HAEMATURIA

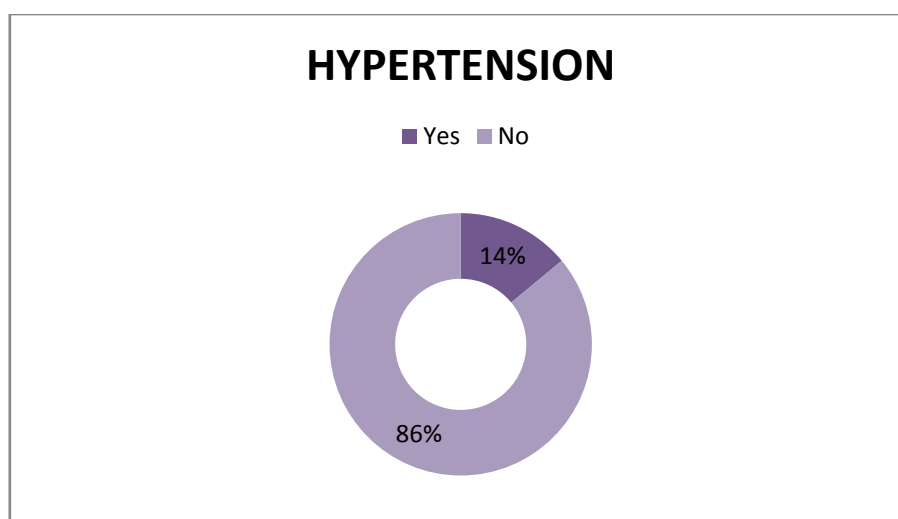
HEMATURIA	FREQUENCY	PERCENT
Yes	10	20.0
No	40	80.0

DYSPNEA

DYSPNEA	FREQUENCY	PERCENT
Yes	4	8.0
No	46	92.0

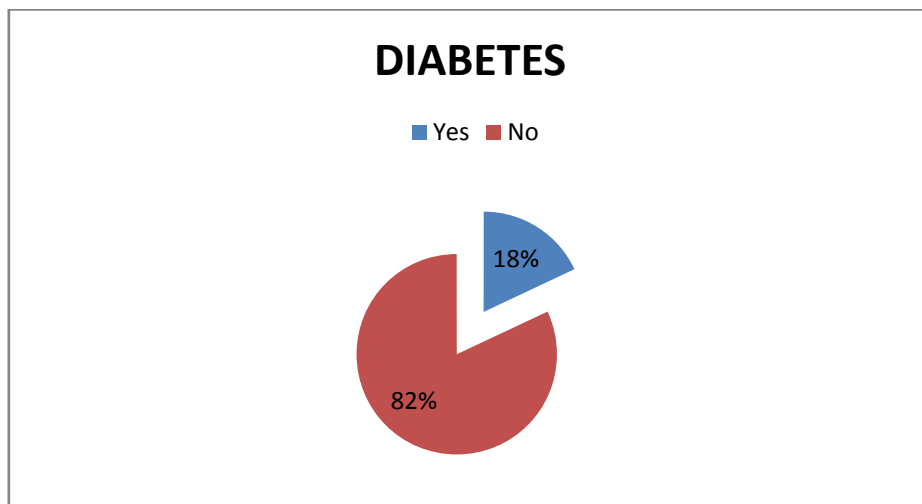
HYPERTENSION

HYPERTENSION	FREQUENCY	PERCENT
Yes	7	14.0
No	43	86.0



DIABETES

DIABETES	FREQUENCY	PERCENT
Yes	9	18.0
No	41	82.0



DYSLIPIDEMIA

DYSLIPIDEMIA	FREQUENCY	PERCENT
Yes	6	12.0
No	44	88.0

CORONARY ARTERY DISEASE

Coronary artery disease	Frequency	Per cent
Yes	5	10.0
No	45	90.0

HYPOTHYROIDISM

HYPOTHYROIDISM	FREQUENCY	PERCENT
Yes	3	6.0
No	47	94.0

SLE

SLE	FREQUENCY	PERCENT
Yes	2	4.0
No	48	96.0

PALLOR

PALLOR	FREQUENCY	PERCENT
Yes	7	14.0
No	43	86.0

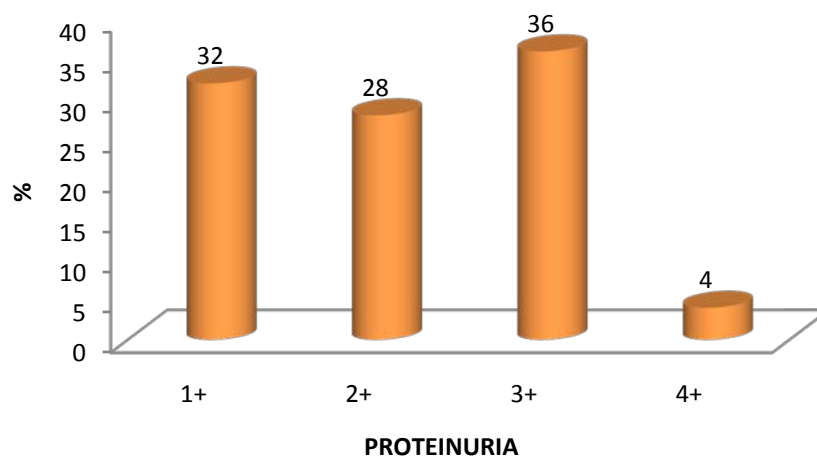
SKIN LESION

SKIN LESION	FREQUENCY	PERCENT
Yes	3	6.0
No	47	94.0

URINE PROTEIN

URINE PROTEIN	FREQUENCY	PERCENT
1+	16	32.0
2+	14	28.0
3+	18	36.0
4+	2	4.0

PROTEINURIA



URINE SUGAR

URINE SUGAR	FREQUENCY	PERCENT
Nil	41	82.0
2+	5	10.0
3+	4	8.0

HIV/HBsAg/AntiHCV

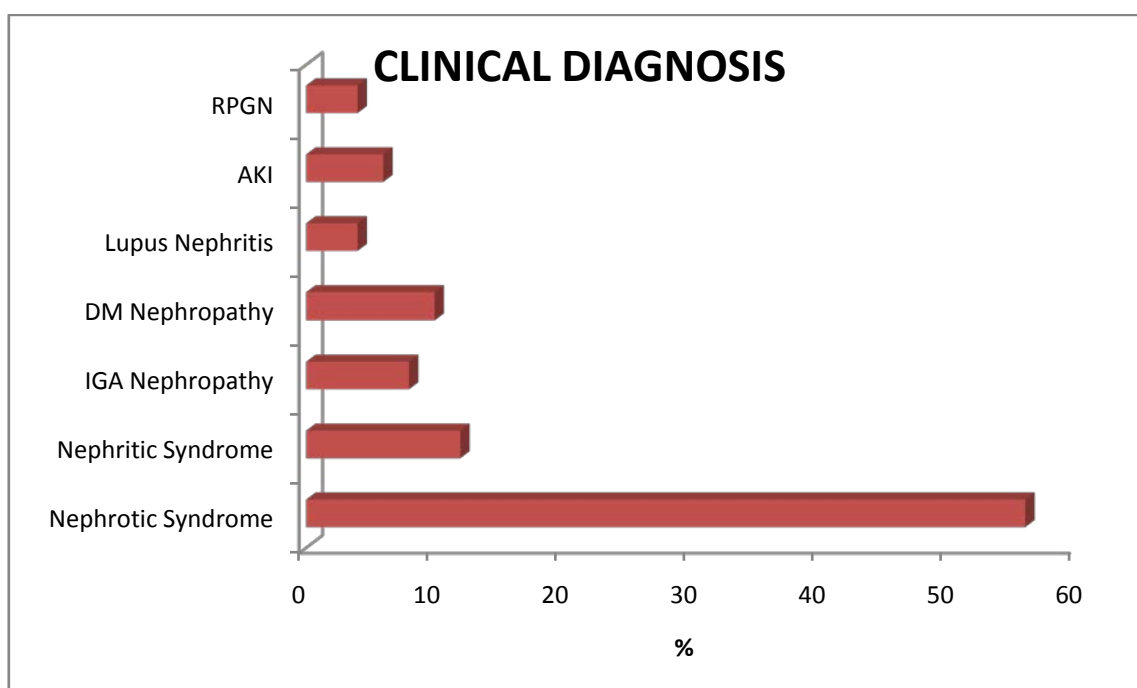
HIV/HBsAg/Anti HCV	Frequency	Per cent
Negative	50	100.0

ANA

ANA	FREQUENCY	PERCENT
Positive	3	6.0
Negative	47	94.0

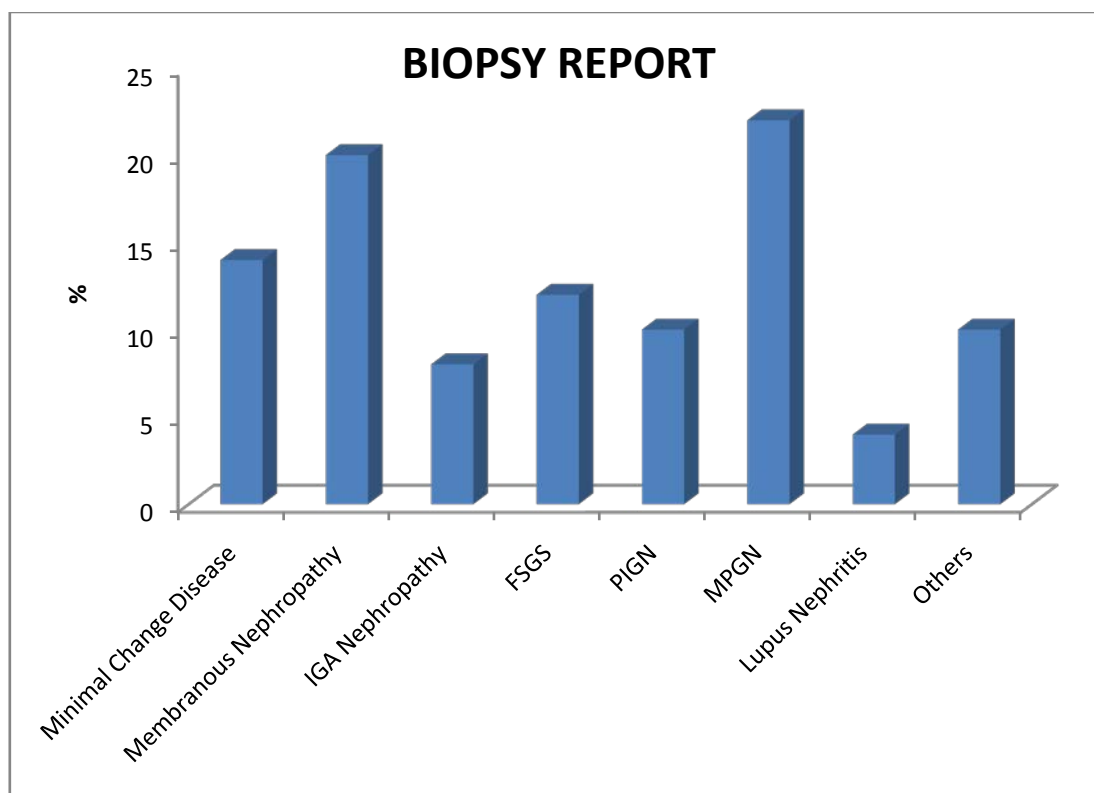
CLINICAL DIAGNOSIS

CLINICAL DIAGNOSIS	FREQUENCY	PERCENT
Nephrotic Syndrome	28	56.0
Nephritic Syndrome	6	12.0
IGA Nephropathy	4	8.0
DM Nephropathy	5	10.0
Lupus Nephritis	2	4.0
AKI	3	6.0
RPGN	2	4.0



BIOPSY REPORT

BIOPSY REPORT	FREQUENCY	PERCENT
Minimal Change Disease	7	14.0
Membranous Nephropathy	10	20.0
IGA Nephropathy	4	8.0
FSGS	6	12.0
PIGN	5	10.0
MPGN	11	22.0
Lupus Nephritis	2	4.0
Others	5	10.0



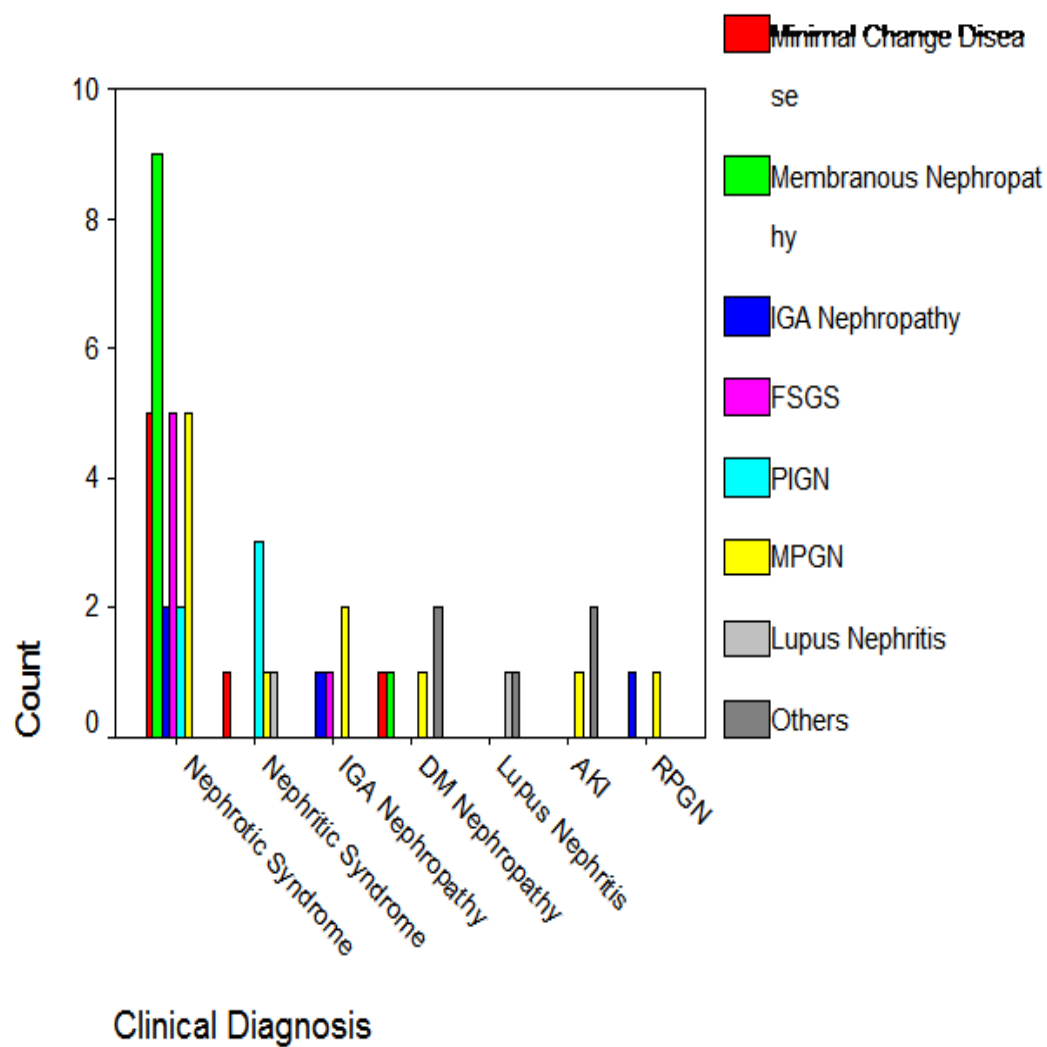
DESCRIPTIVE STATISTICS

VARIABLE	MIN	MAX	MEAN	STD. DEVIATION
Age in years	14	69	35.14	13.337
SBP	100	180	137.20	20.508
DBP	60	110	85.40	12.651
Hb	6.4	14.4	10.882	1.9355
B.Urea	48	106	65.98	12.091
S.Creatinine	1.1	14.1	2.684	1.8679
Sr.Cholesterol	146	341	188.36	41.364
TGL	96	172	114.84	14.933

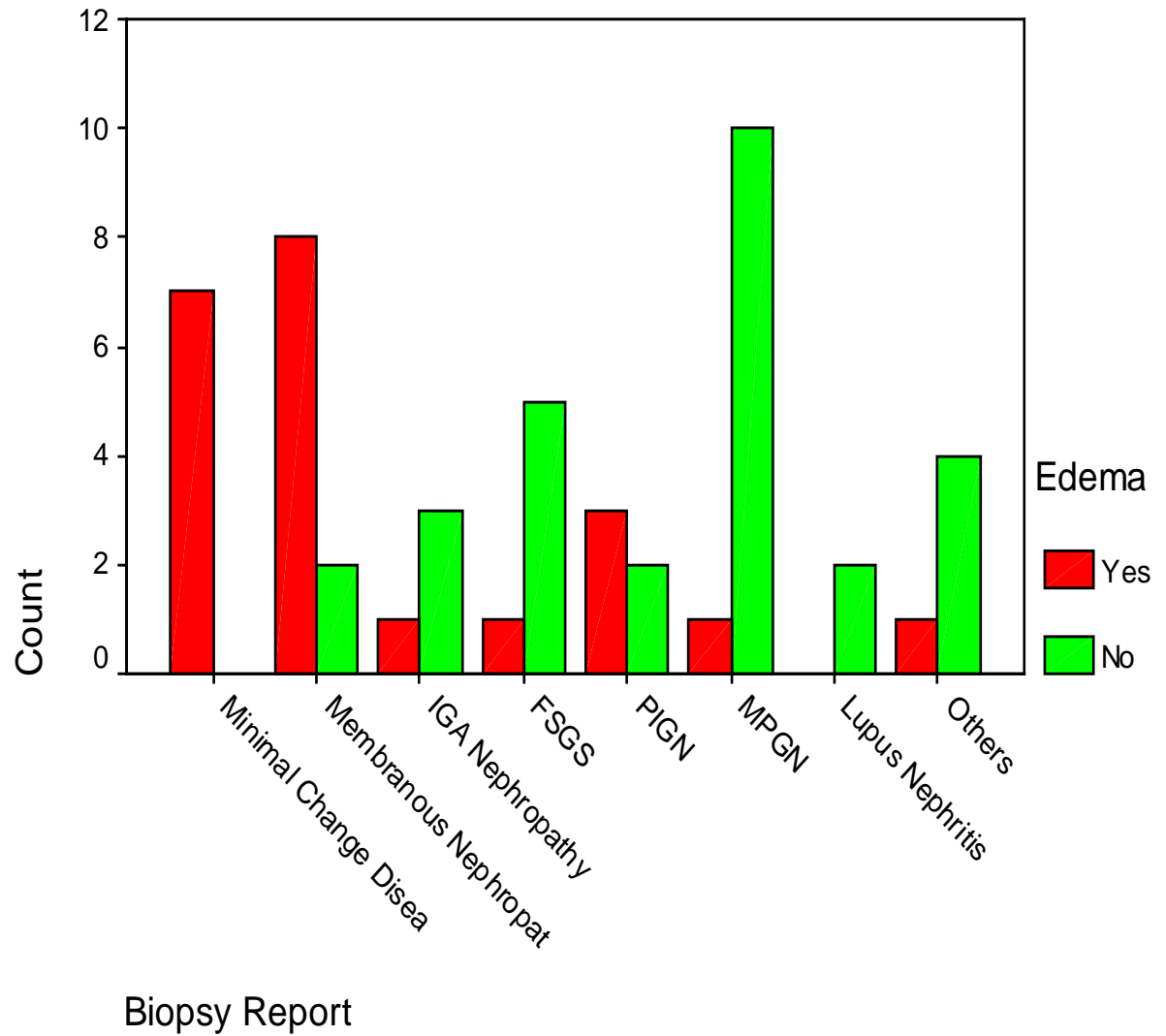
Clinical Diagnosis VS Biopsy Report

Clinical Diagnosis	Biopsy Report							
	Minimal Change Disease	Membranous	IGA Nephropathy	FSGS	PIGN	MPGN	Lupus	Others
Nephrotic Syndrome	5	9	2	5	2	5	0	0
Nephritic Syndrome	1	0	0	0	3	1	1	0
IGA Nephropathy	0	0	1	1	0	2	0	0
DM Nephropathy	1	1	0	0	0	1	0	2
Lupus Nephritis	0	0	0	0	0	0	1	1
AKI	0	0	0	0	0	1	0	2
RPGN	0	0	1	0	0	1	0	0

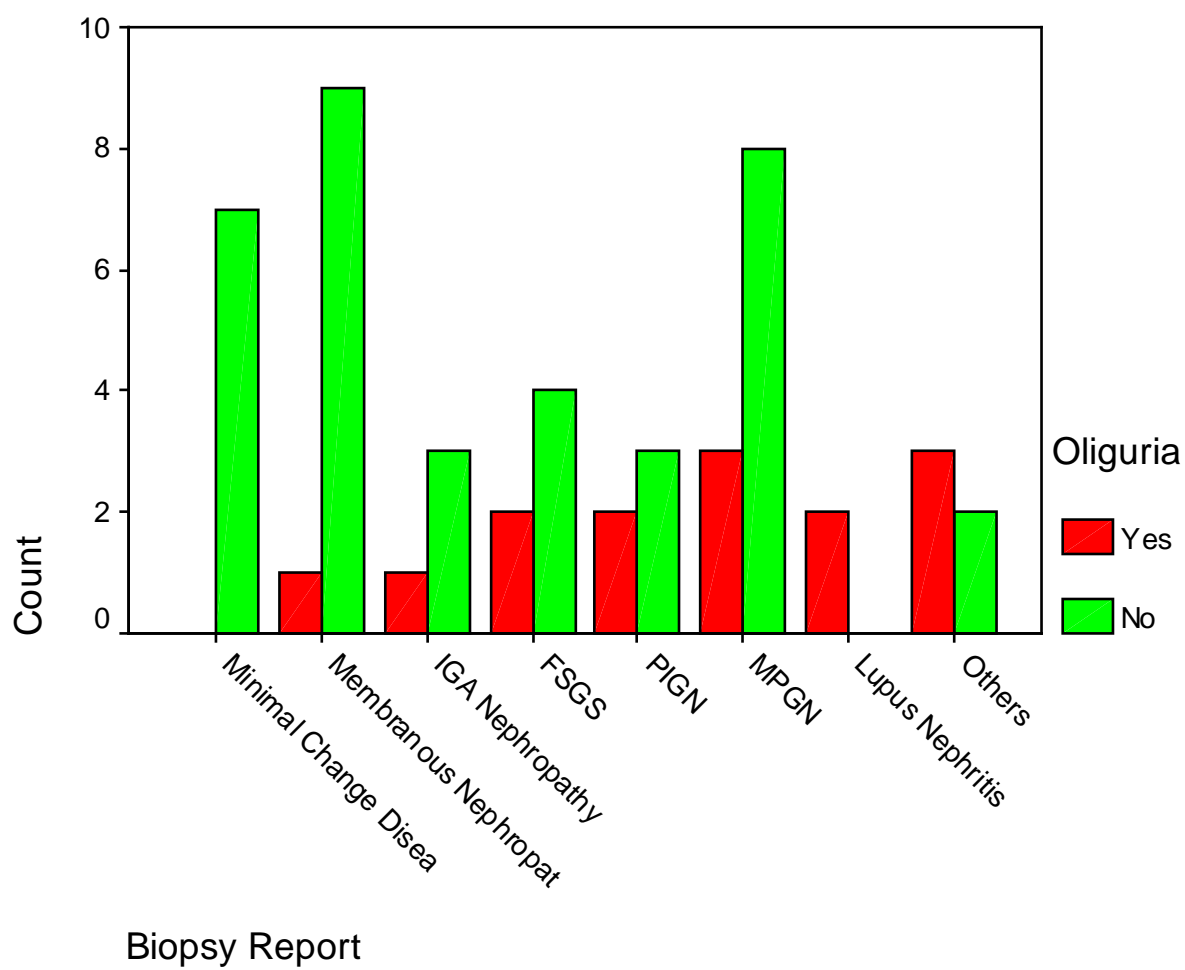
CLINICAL DIAGNOSIS VS BIOPSY REPORT



Biopsy Report VS Edema

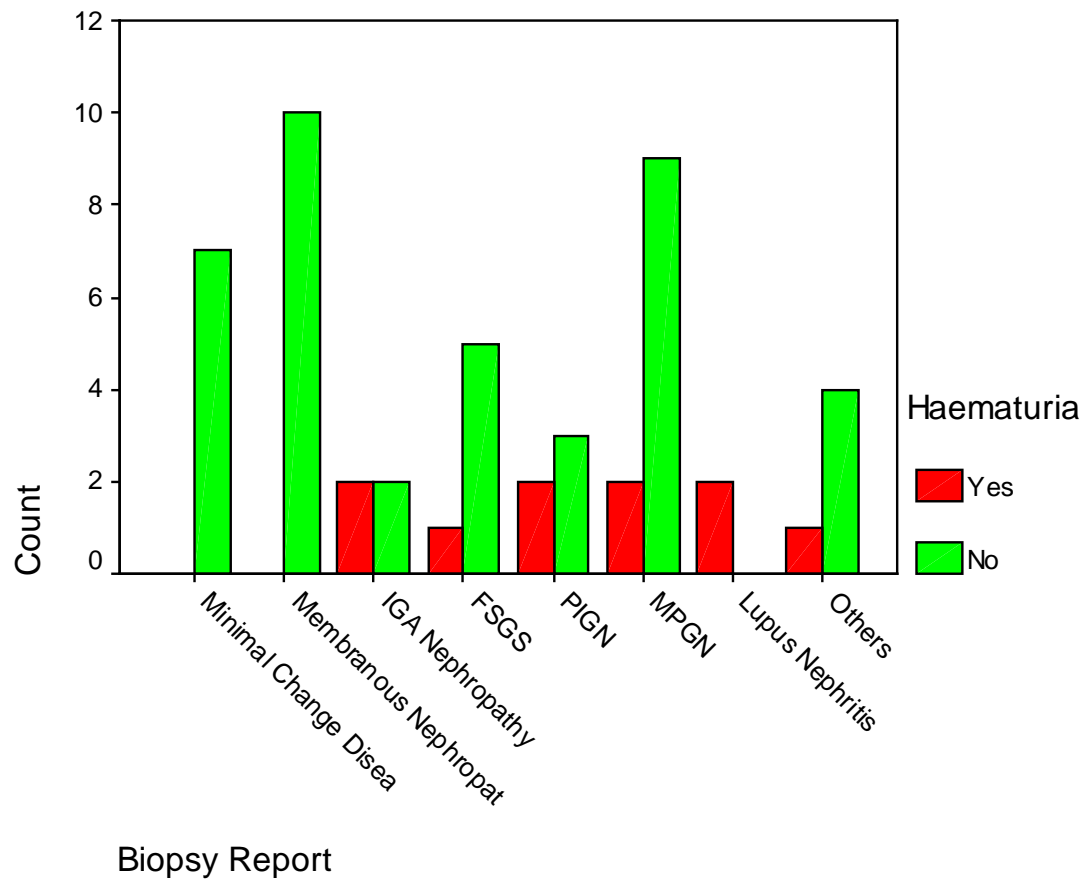


Biopsy Report VS Oliguria

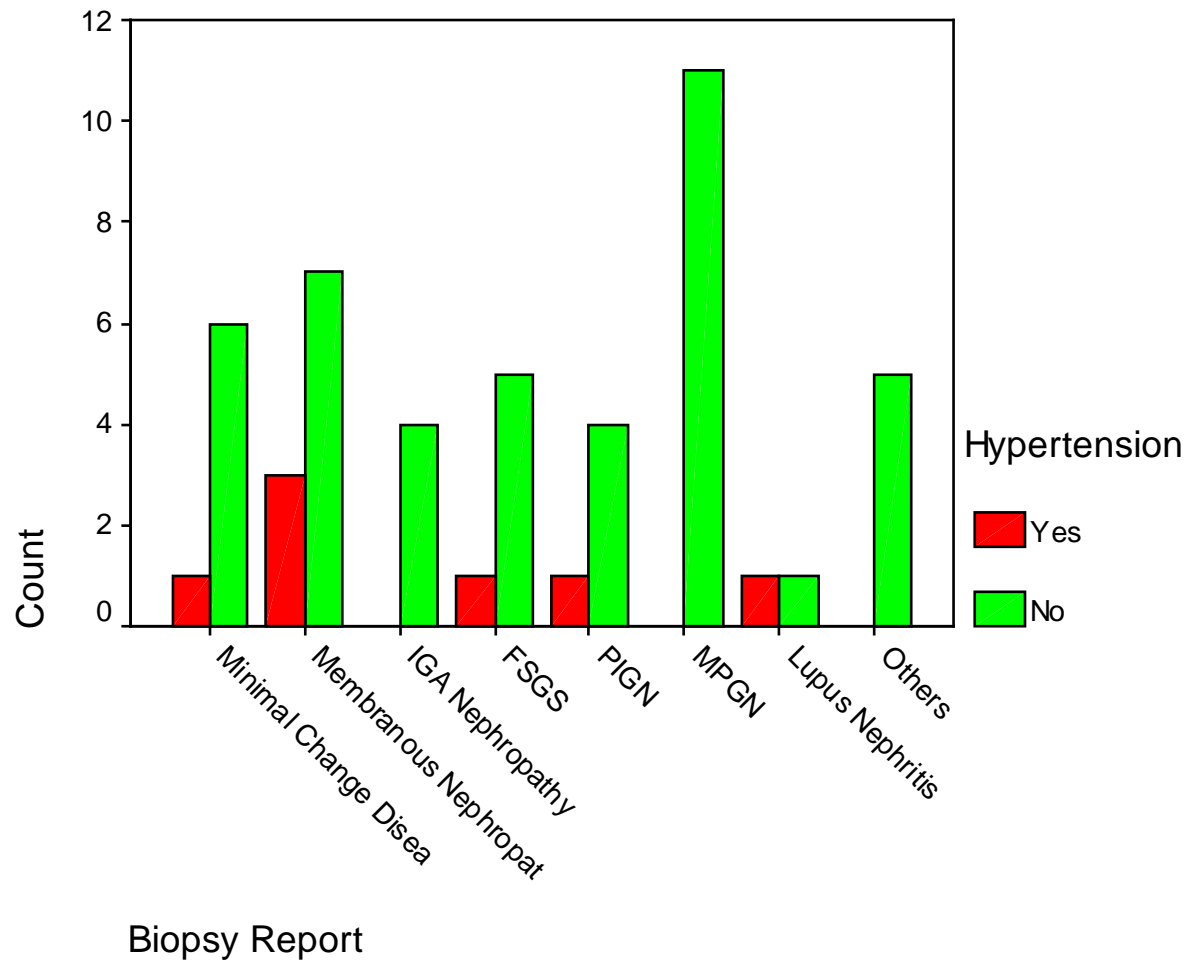


Biopsy Report VS Haematuria

Patients with IGA nephropathy, FSGS, PIGN, MPGN and all patients with lupus nephritis had hematuria.

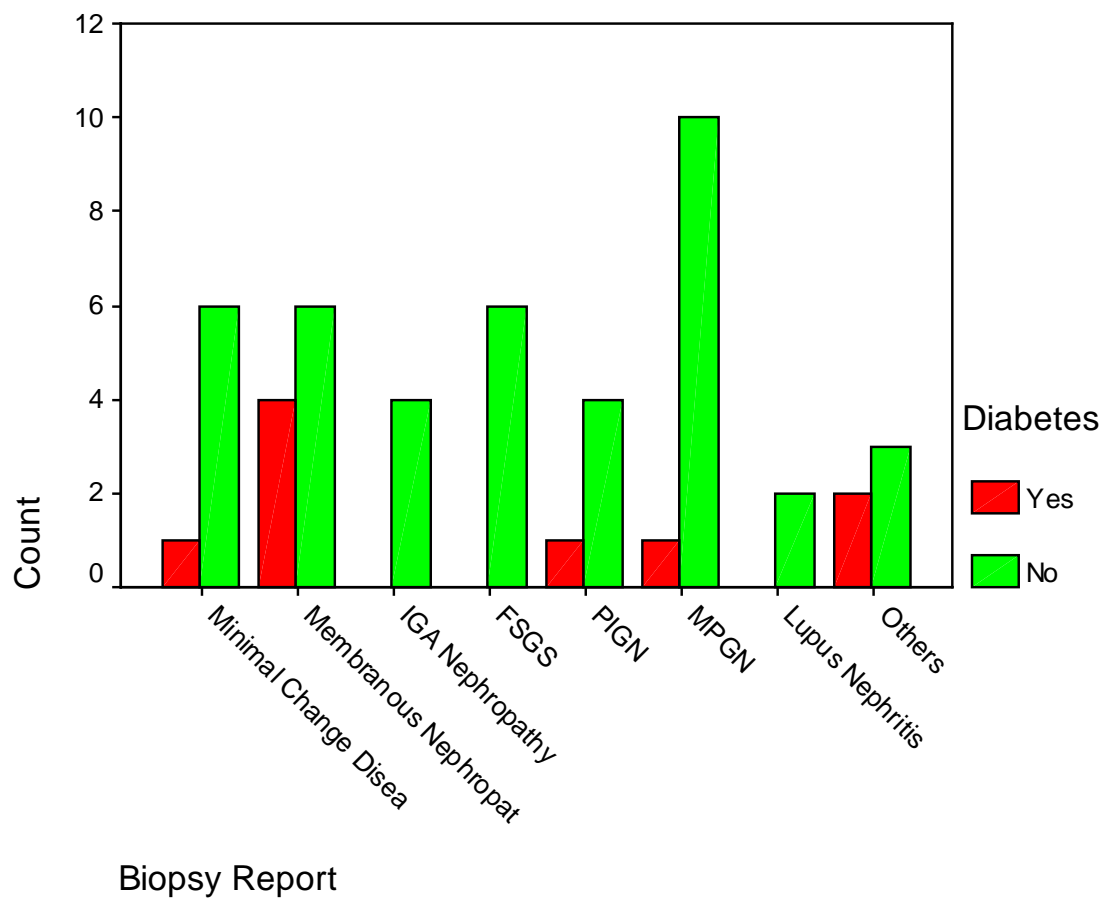


Biopsy Report VS Hypertension

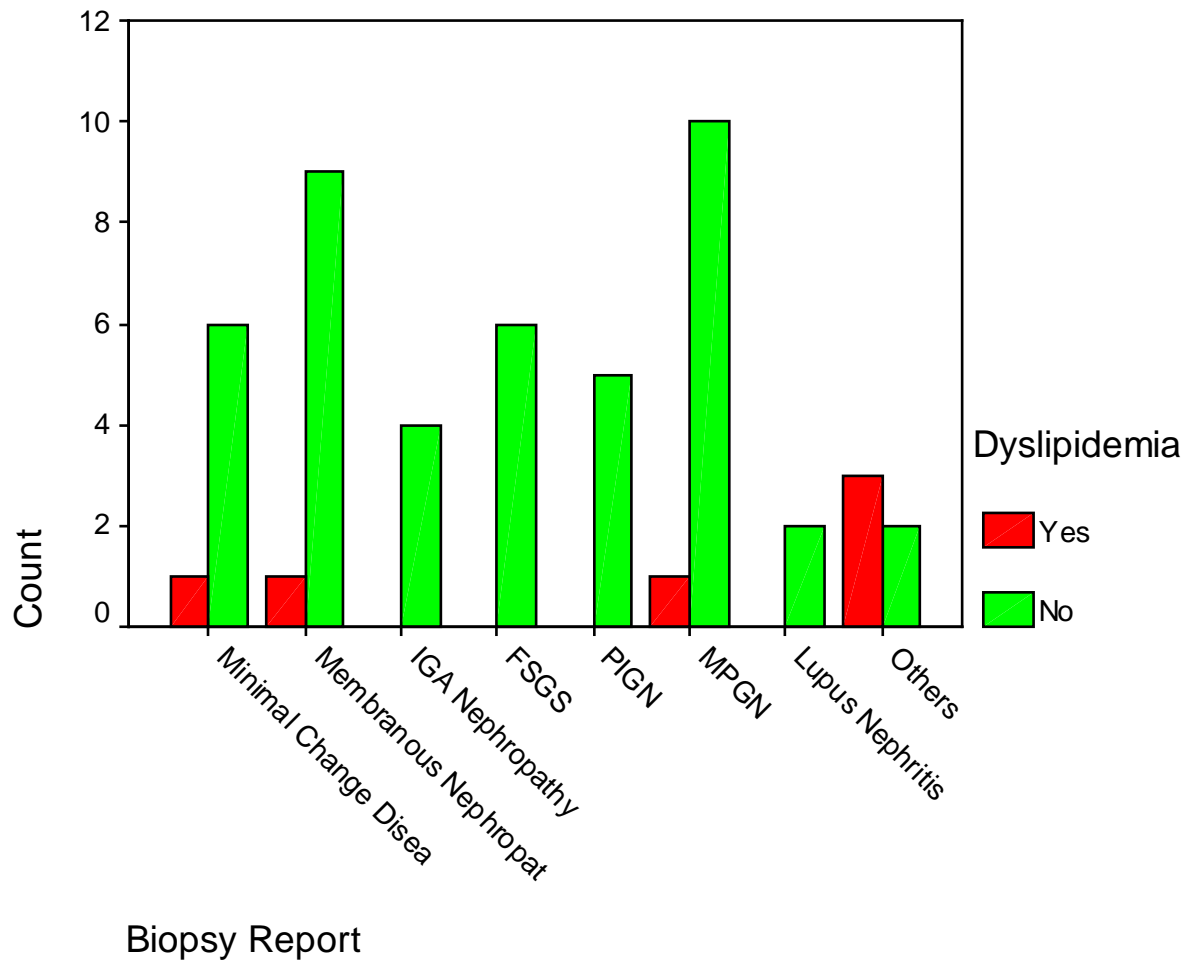


Biopsy Report VS Diabetes

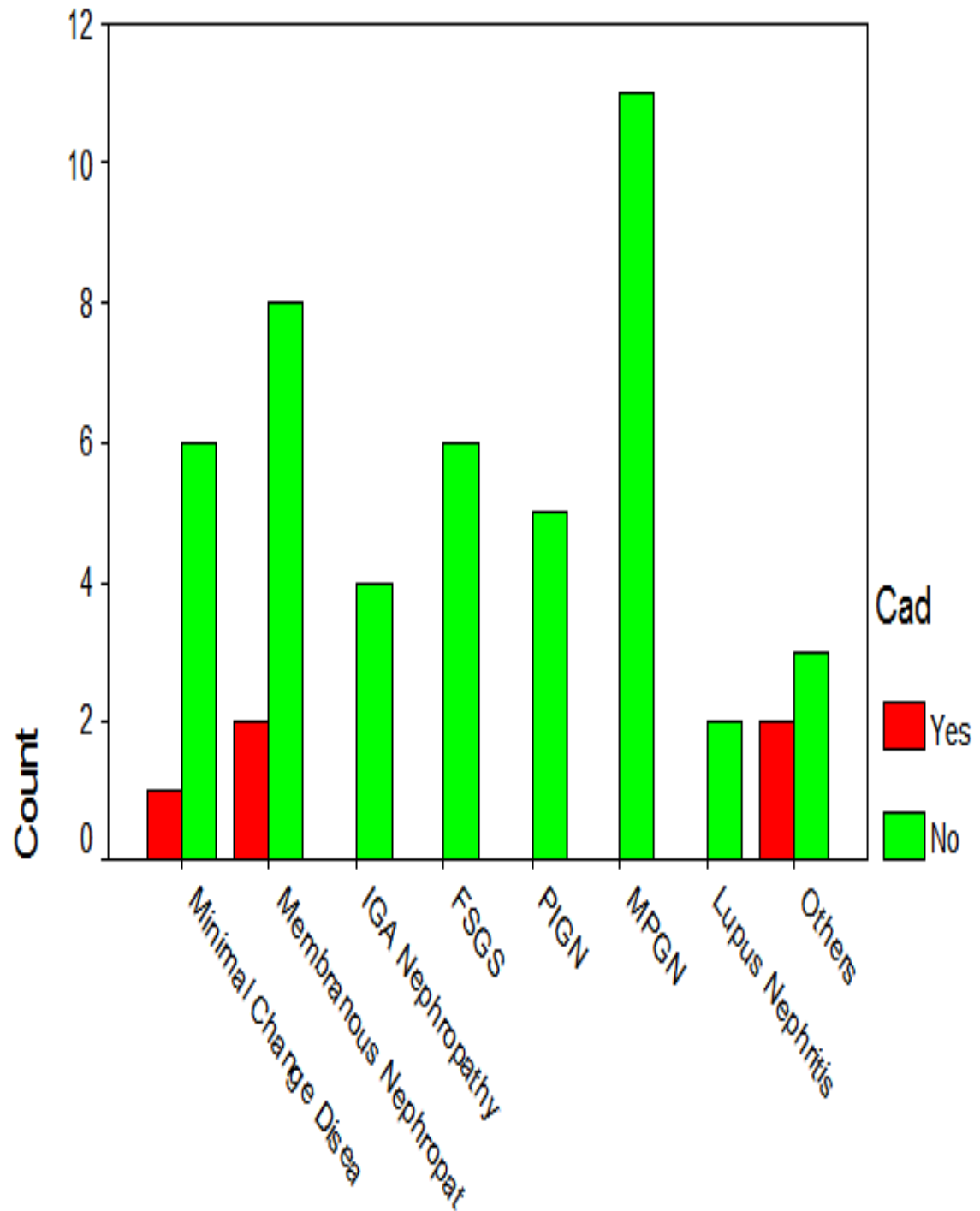
Patients with diabetes mostly had membranous nephropathy and also MCD, MPGN.



Biopsy Report VS Dyslipidemia

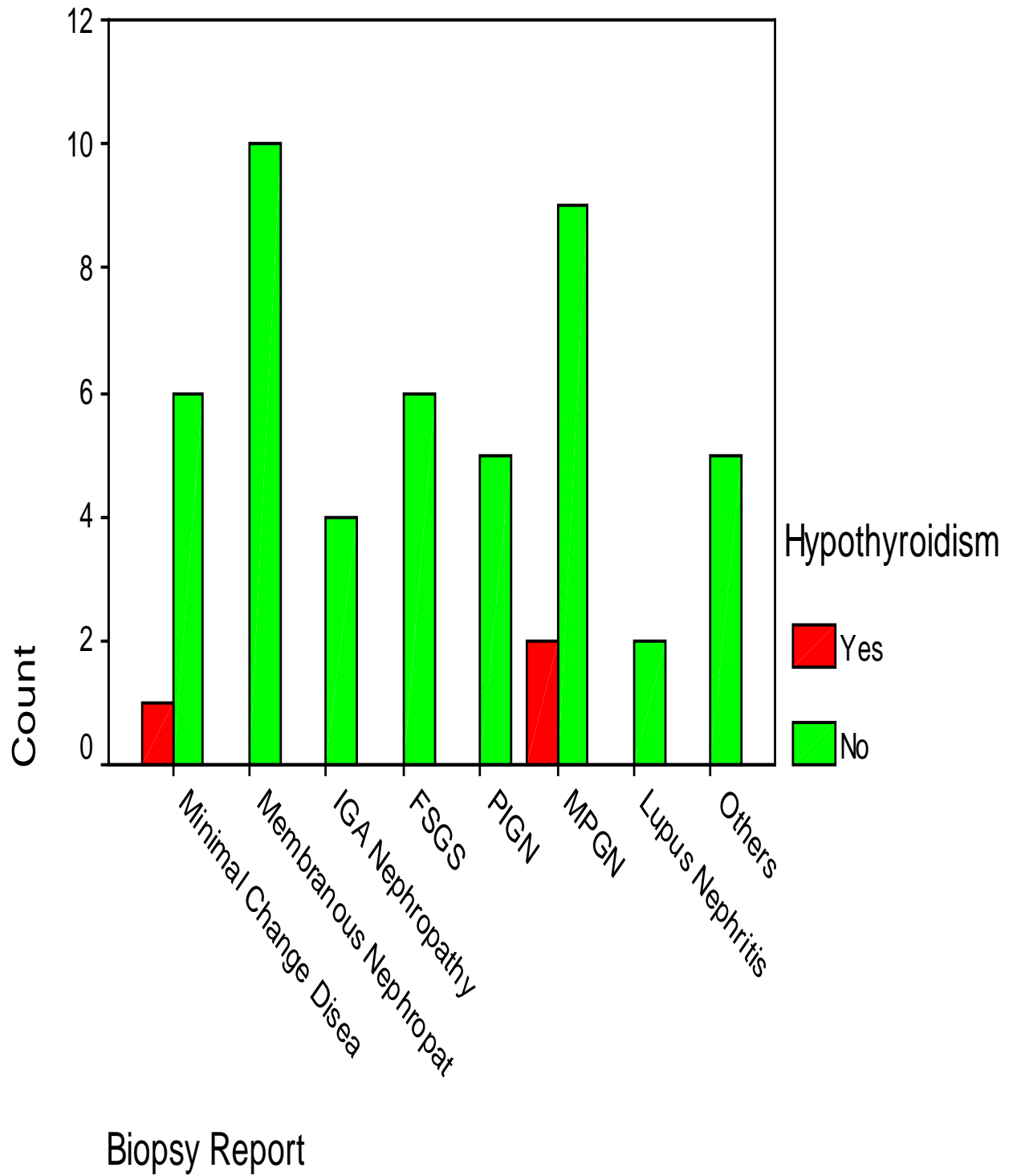


Biopsy Report VSCAD



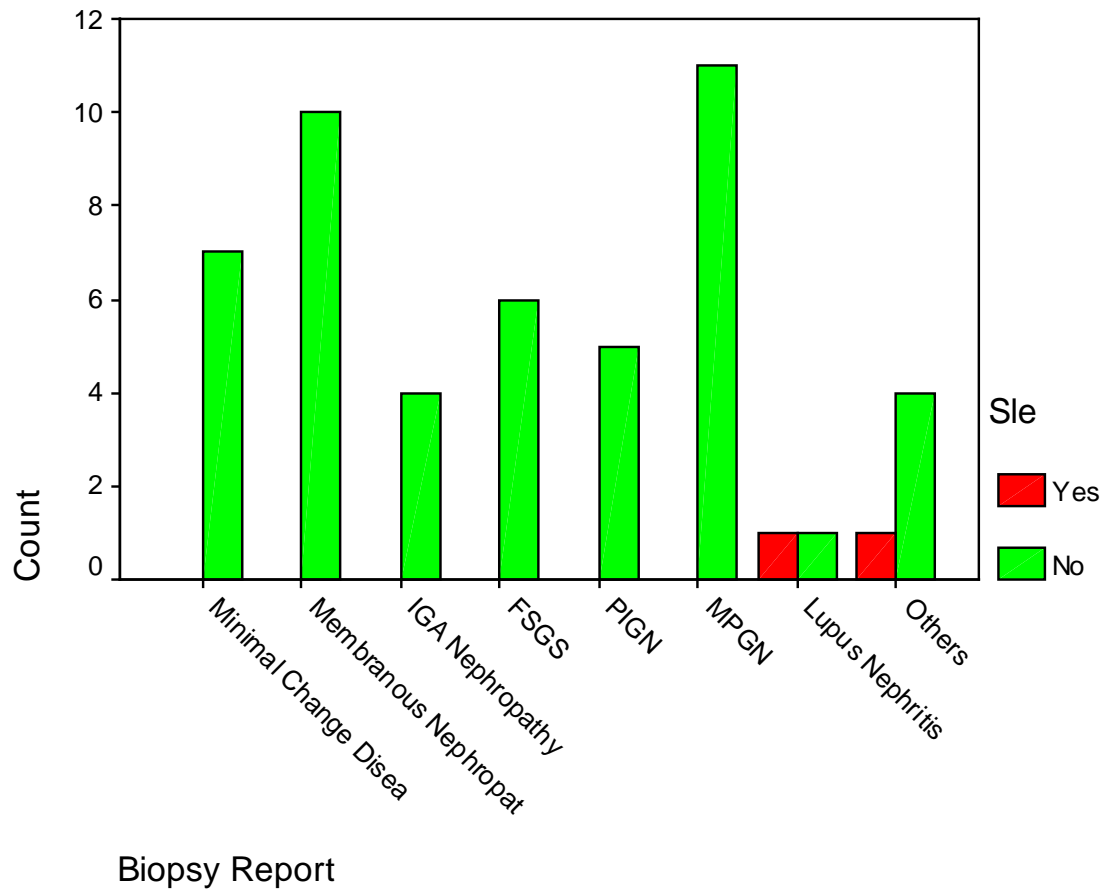
Biopsy Report

Biopsy Report VS Hypothyroidism



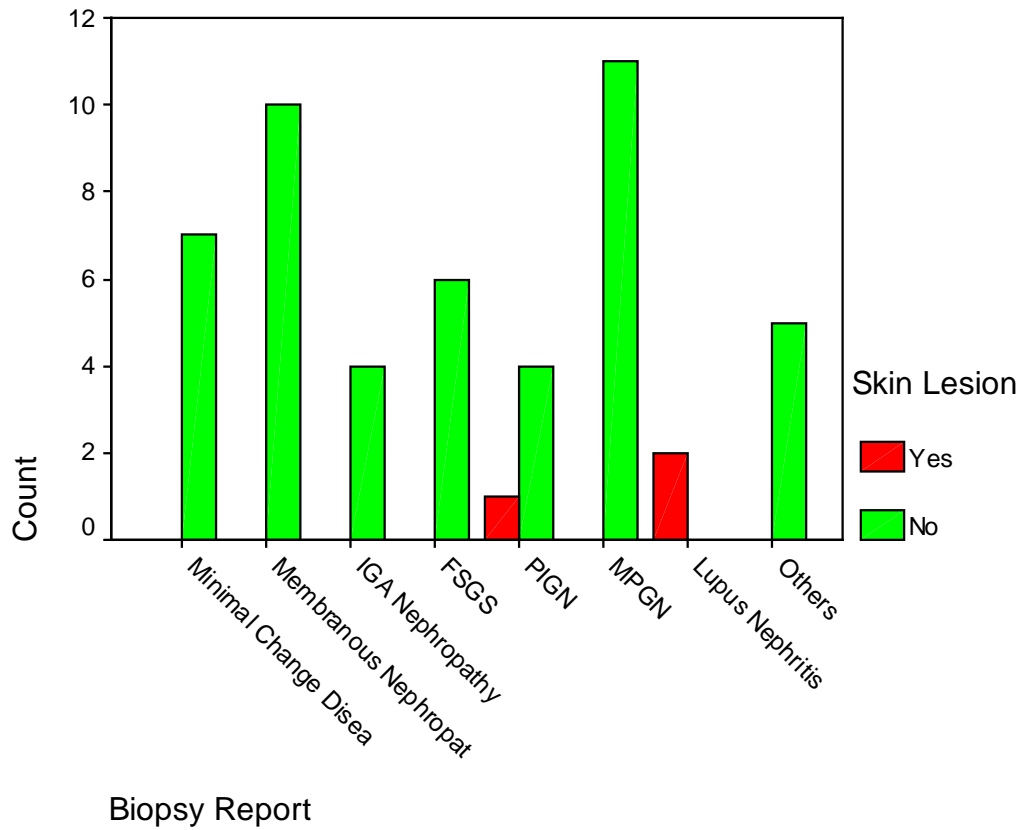
Biopsy Report VS SLE

All patients with SLE had LUPUS NEPHRITIS in their renal biopsy.

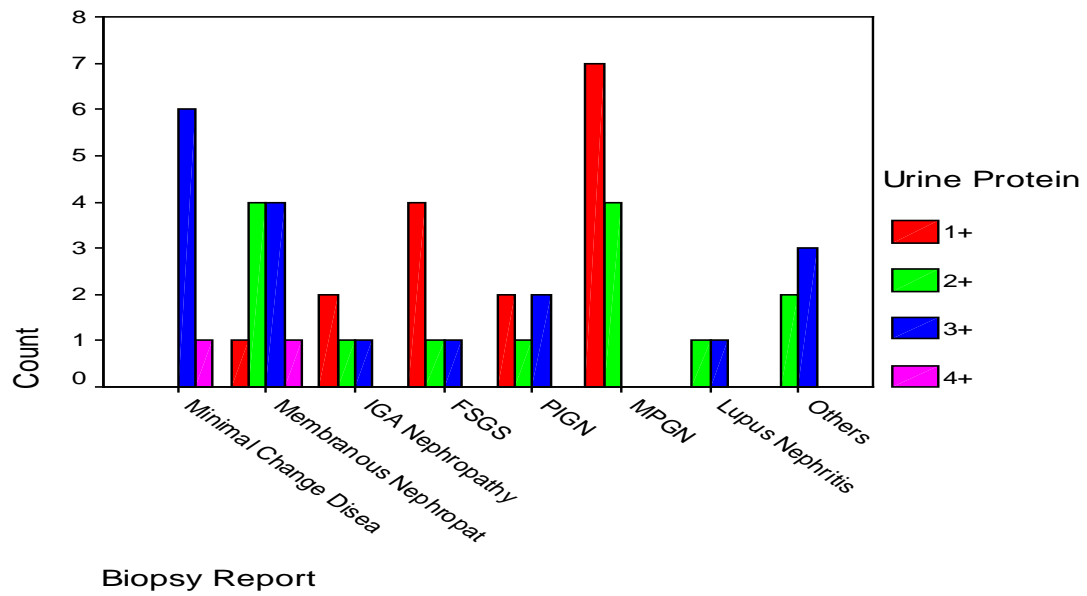


Biopsy Report VS Skin Lesion

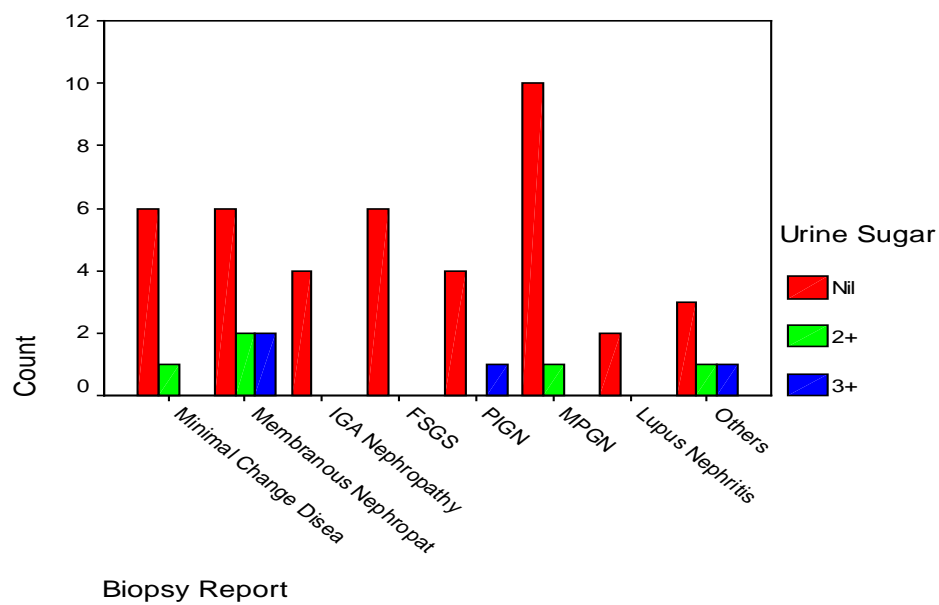
Patients with skin lesion had either PIGN or SLE.



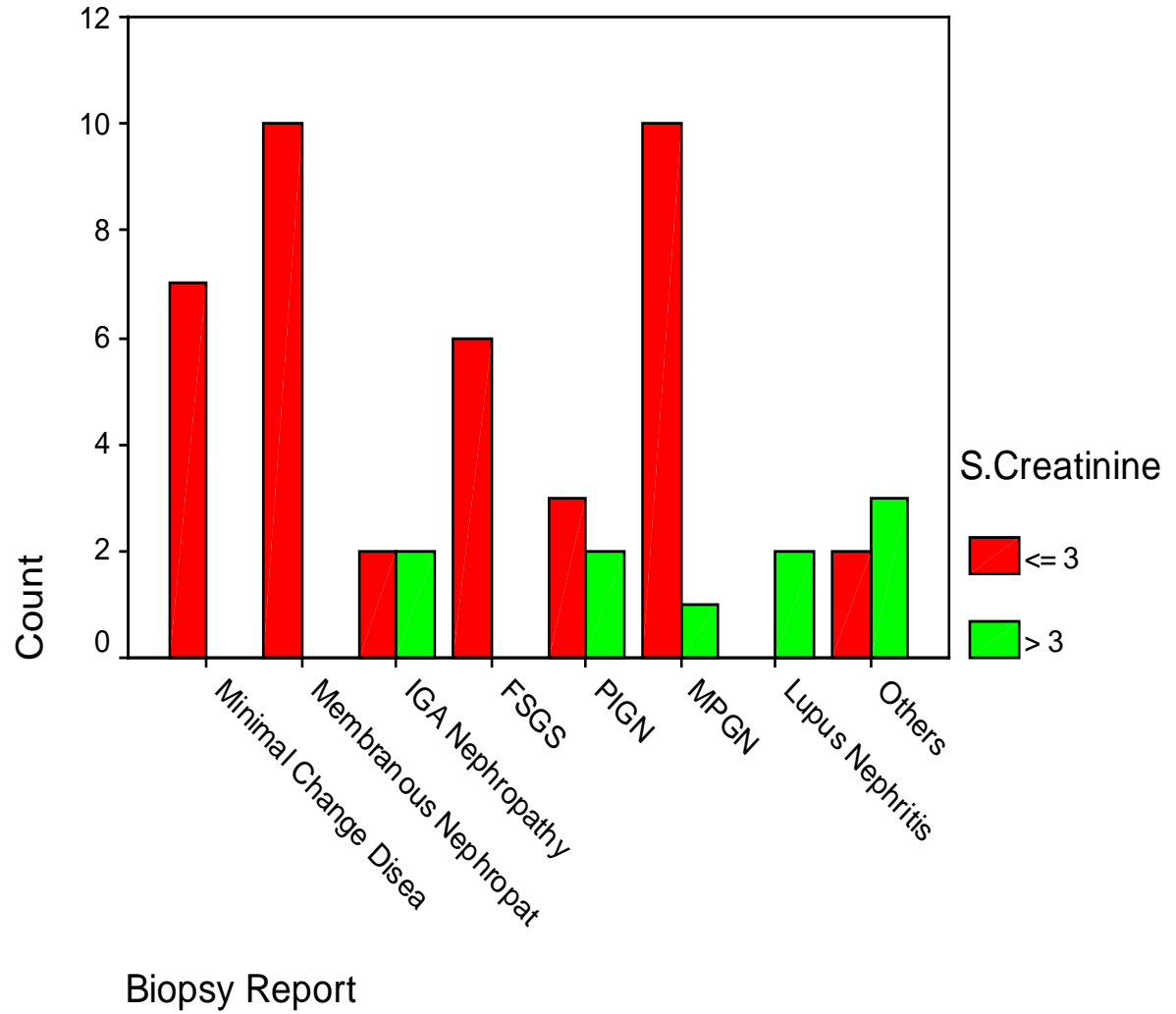
Biopsy Report VS Urine Protein



Biopsy Report VS Urine Sugar

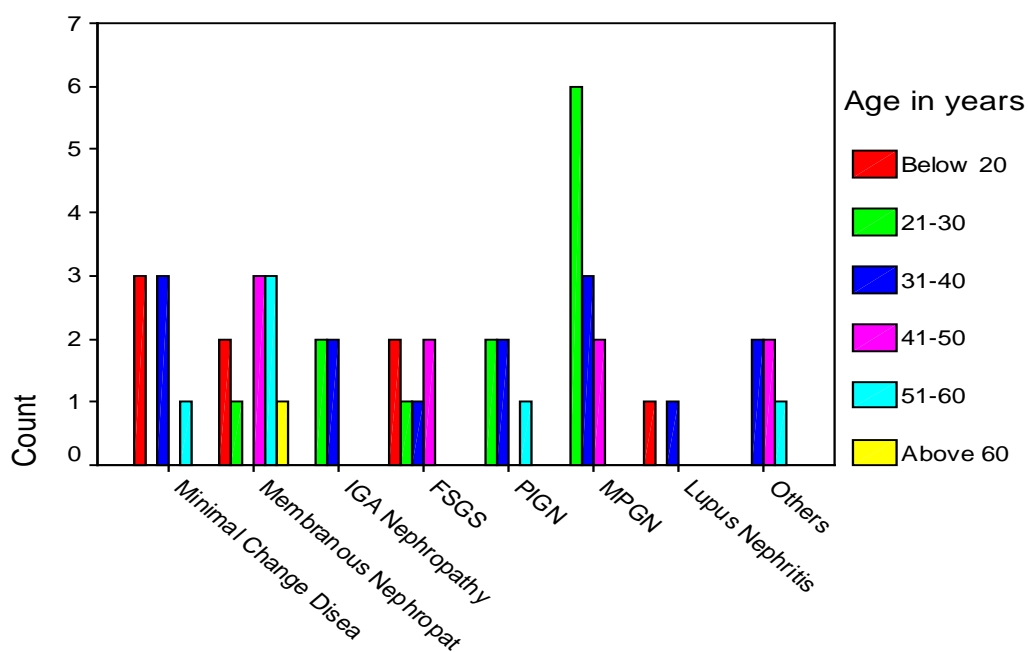


Biopsy Report VS S.Creatinine



Biopsy Report VS Age in years

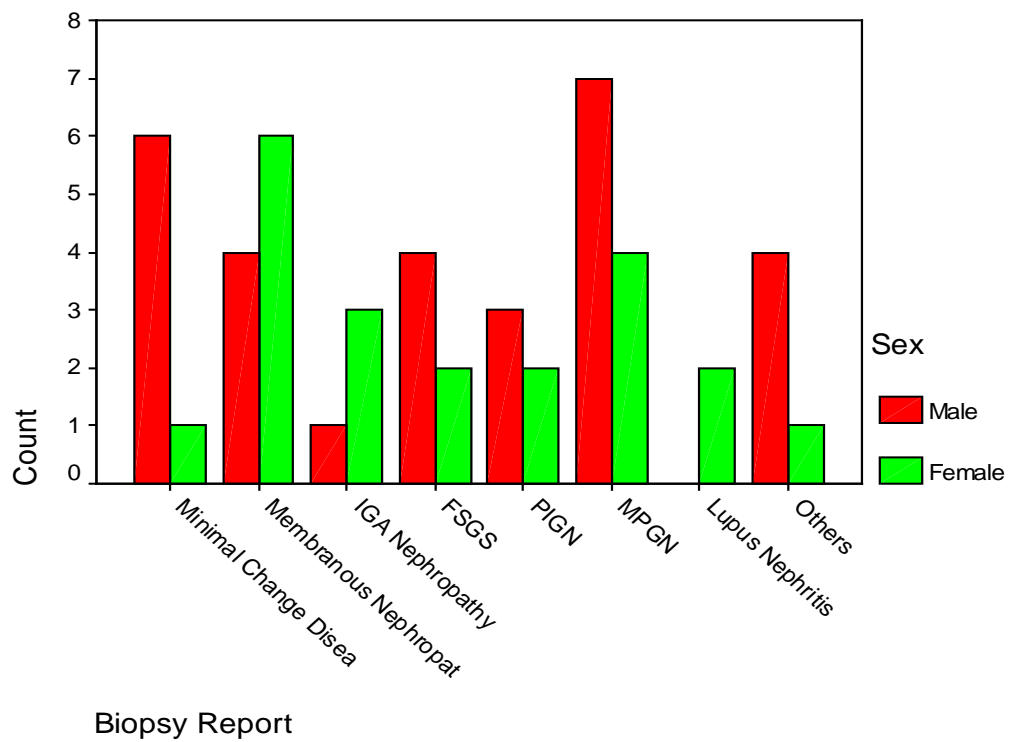
BIOPSY	AGE IN YEARS					
	< 20	21-30	31-40	41-50	51-60	>60
Minimal Change Disease	3	0	3	0	1	0
Membranous Nephropathy	2	1	0	3	3	1
IGA Nephropathy	0	2	2	0	0	0
FSGS	2	1	1	2	0	0
PIGN	0	2	2	0	1	0
MPGN	0	6	3	2	0	0
Lupus Nephritis	1	0	1	0	0	0
Others	0	0	2	2	1	0



Biopsy Report

Biopsy Report VS Sex

BIOPSY REPORT	SEX	
	MALE	FEMALE
Minimal Change Disease	6	1
Membranous Nephropathy	4	6
IGA Nephropathy	1	3
FSGS	4	2
PIGN	3	2
MPGN	7	4
Lupus Nephritis	0	2
Others	4	1



DISCUSSION

DISCUSSION

Glomerular diseases are an important cause of end-stage kidney disease. The histologic spectrum of these is different in adults as compared with children as well as in tropical as compared with temperate countries. In the present study, primary glomerular diseases accounted for 89% cases of nephrotic syndrome, while LN was the most common secondary cause. Overall, membranous nephropathy was the most common cause of nephrotic syndrome.

An analysis of the spectrum of primary glomerular diseases as a cause of nephrotic syndrome during the last five decades has revealed a 5-fold increase in the frequency of FSGS. Along with that, there was a 3-fold increase in the frequency of MGN making it the second most cause of nephrotic syndrome while DPGN decreased to one-tenth of its earlier prevalence. There was no significant change in MCD or MPGN^[48,49,50]. This trend is similar to the emerging global trend, which indicates an increase in the incidence of FSGS making it the number one cause of nephrotic syndrome world-wide. There can be a variety of reasons for this changing spectrum. This may be related to improvement in the overall quality-of-life, decreased rate of infections, better socio-economic status, increased incidence of obesity and changing pattern of indications for renal biopsy.

A more widespread use of IF and electron microscopy in the analysis of renal biopsy can explain increased diagnosis of MGN and FSGS, which are otherwise likely to be misdiagnosed as MCD.

While the earlier studies found MCD to be the most common cause, more recent ones show results similar to our study. The study done from Vellore in 1970's noted that MCD accounted for about 35% of all cases of nephrotic syndrome. Similarly, studies from Delhi and Rohtak, also found MCD to be responsible for more than one-third of nephrotic syndrome^[51,52] The study done at Vellore in 1990's found that the incidence of FSGS had increased from 15% to 19% and it became the most common underlying etiology for primary nephrotic syndrome.

In a recent study published from Kolkata, Golay *et al.*, found that FSGS was underlying disease in 27.4% of their patients making it the most common one while MGN was third most common accounting for about 25%. This figure is not similar to our present data, where FSGS and MGN were responsible for 12% and 20% of cases respectively. However, they found MCD in 27.1% of cases, making it the second most common cause of nephrotic syndrome while MPGN was seen in only 18%. This is similar to our data where MPGN was seen in about 16% of cases while MCD was seen in around 15% of cases. The

exact reason for this difference is not clear. A possible explanation may be that only 5.6% of patients in their study were subjected to EM examination in contrast to about one-fourth in our study.

In a study published by Siegel *et al.*, it was observed that EM is essential for correct diagnosis in 11% of cases and for confirmation of diagnosis in an additional 36%.^[53] The incidence of IgAN was also less at 8% in our study as compared to 4-14% in other studies from India probably since the majority of those with IgAN do not have a nephrotic syndrome.

The data from the West are also conflicting. Studies done in USA have clearly demonstrated increasing incidence of FSGS particularly in African-Americans making it the most common cause of nephrotic syndrome in their adult population. Not only this, the proportion of FSGS as cause of end-stage renal disease in USA has increased almost ten times in the last two decades.^[54] Similarly, studies done from some other parts of the world have shown FSGS to be the most common cause of adult nephrotic syndrome.^[55,56]

There is emerging evidence that its incidence in children is also increasing and a study done in Indian pediatric patients has demonstrated FSGS to be the most common cause in adolescents as compared with MCD in younger patients.^[57] However, the data from some European countries including registry

data do not agree with this trend. Studies done from Italy and Spain have shown MGN to be the most common cause of adult nephrotic syndrome^[58,59] while those from Denmark, the Czech republic and Romania have shown MCD, IgAN and MPGN respectively to be the most common lesions^[60,61,62]

While studies from Pakistan and Nepal have shown that IgAN is an infrequent cause of nephrotic syndrome with figures of around 2% the ones from China and Korea have found it to be very common. Changet *et al.*, observed that IgAN was responsible for 28.3% of nephrotic syndrome making it the most common cause in Korea. Zhou *et al.*, found IgAN to be the second most common cause of nephrotic syndrome after MGN, accounting for 20% of cases in China. However, Kazi *et al.*, from Pakistan have found FSGS to be the most common cause accounting for almost 40% of their cases, followed by MGN (26.6%) and MCD (14.8%).

In 2006, Nair and walker after studying the 5 years results of renal biopsy, they reported that IgA nephropathy is the commonest primary glomerular disorder in United states and also in mid-western region. But focal segmental glomerulosclerosis is the commonest one in African Americans. Same results are reported by swaminathan et al in Minnesota, which explained

the order of prevalence as following; IgA nephropathy, focal segmental glomerulosclerosis and membranous nephropathy.

A cross sectional study conducted by politico et al in Brazil reported the focal segmental glomerulosclerosis as most common one following which membranous nephropathy and IgA nephropathy respectively. He also mentioned that most common secondary glomerulopathy is lupus nephritis and next common is post infectious glomerulopathy.

In Europe the most common primary glomerulopathy is IgA nephropathy and the most common secondary glomerulopathy is lupus nephritis. The Asian picture also denotes the same. But in Middle East the commonest form is focal segmental glomerulopathy where the IgA nephropathy is only 6.5%. But most common secondary form is lupus nephritis as like as Europe. The Australian data also explains that IgA is the commonest one in their country.

Out of 50 patients we had 8 patients under the age of 20, 12 patients between the age group of 21-30, 14 patients between the age group of 31-40, 9 patients between the age group of 41-50, 6 patients above the age of 51. Out of 50 patients 16 were smoker and 14 were alcoholic. 22 patients presented with edema, 14 patients presented with oliguria, 10 patients presented with hematuria

and 4 presented with dyspnoea. 7 patients were known hypertensive, 9 were diabetic, 6 were dyslipidemic, 5 patients had ischemic heart disease, 3 had hypothyroidism and 2 were suspected as SLE. On examination pallor found in 7 patients, skin lesion found in 3 patients.

On laboratory investigation urine protein found 1+ in 16 patients, 2+ in 14 patients, 3+ in 18 patients and 4+ in 2 patients. Urine sugar was found positive in 9 patients and negative in 41 patients. HBsAg, Anti-HCV and HIV were negative in all patients. ANA was found positive in 3 patients. The most common clinical diagnosis encountered in our study is nephrotic syndrome, next one is nephritic syndrome. IgA nephropathy was the clinical diagnosis in 4 patients, Diabetic nephropathy in 5 patients, Lupus nephritis in 2 patients, acute kidney injury in 3 patients and RPGN in 2 patients.

We had significant abnormalities in urine examination like granular casts, RBC casts and high proteins. Systolic blood pressure was found above 140 in 28 patients and diastolic blood pressure was found above 90 in 27 patients. Haemoglobin was found less than 10 gm% in 15 patients and more than 10 in other patients. Lowest creatinine value found was 1.1mg% and highest was 14.1mg%.

After renal biopsy, the most common types of histopathological diagnosis we had obtained are minimal change nephropathy and membranous nephropathy. IgA nephropathy was found in 4 patients, Focal segmental glomerulosclerosis was found in 6 patients, Post infectious glomerulonephritis was found in 5 patients, Membranoproliferative glomerulonephritis was found in 7 patients, Lupus nephritis was found in 2 patients and other categories were found in 5 patients which included thrombotic microangiopathy, crescentic nephritis and cast nephropathy. The P value of correlation of clinical and histopathological diagnosis is 0.006 and highly significant.

Correlation of edema and histopathological diagnosis gave the P value of significant level. Correlation of hematuria and histopathological diagnosis was also found significant as the P value is 0.027. Correlating other symptoms like oliguria was found low significant. We also correlated the co-morbidities like hypertension, diabetes, dyslipidemia, coronary artery disease, hypothyroidism and systemic lupus erythematosus with the biopsy result. Correlation of skin lesion with histopathological diagnosis is found to be highly significant.

In age distribution it is found that membranoproliferative glomerulonephritis appears to be common in third decade of the life, membranous nephropathy appears to be common in fourth and fifth decade, post infectious and IgA nephropathy are appears to common in middle age group, minimal change nephropathy commonly found in under twenties and fourth decade, lupus nephritis appeared mostly in under forties.

In sex distribution it is found that 70-80% of minimal change disease is seen in male whereas almost all lupus nephritis is seen in female. Focal segmental glomerulosclerosis, post infectious glomerulonephritis and membranoproliferative glomerulonephritis are more common in males than females. But IgA nephropathy and membranous nephropathy are more common in females than males. Other uncommon results like cast nephropathy, thrombotic microangiopathy and crescentic glomerulonephritis are found common in males.

We had an interesting patient, who presented with acute renal failure and hypercalcemia. We evaluated that patient, he had multiple lytic bone lesions and myeloma picture in marrow biopsy and finally we identified cast nephropathy which was the cause for his renal failure.

CONCLUSION

Our study revealed that some patients, who clinically presented with nephritic syndrome, were later diagnosed as nephrotic Syndrome after histopathological report of renal biopsy. Likewise some patients, who clinically presented with nephrotic syndrome, were later diagnosed as nephritic syndrome after renal biopsy report. Some of the clinically suspected acute kidney injury was also later diagnosed as chronic glomerular diseases after HPE report. These results show that the diagnosis based on the clinical presentation differs from histopathological diagnosis in many circumstances. So it will be more useful if we get a histopathological diagnosis before concluding the diagnosis and before starting the treatment, whenever needed.

As I mentioned earlier IgA nephropathy is the commonest primary glomerulopathy in most part of the world according to various reports. But our study showed that membranous nephropathy as the commonest one and minimal change disease as second most common. Though the MPGN appears common one it is not true, because for the purpose statistical analysis we included the membranoproliferative, mesangioproliferative and diffuse proliferative under the category of MPGN. In the category of other we included the rare forms like diabetic nephropathy, cast nephropathy, acute tubular necrosis and thrombotic microangiopathy.

A cross sectional study of Italian registry of renal biopsy revealed that most common indication for renal biopsy is clinically diagnosed nephritic syndrome and second most common indication is urinary abnormalities. Hanco et al in United Kingdom reported about the changing pattern of the primary glomerular diseases and also the increasing rate of biopsy from 2.02 to 7.08 per hundred thousand populations per year. These data emphasises the importance of the maintenance of the national level and state level renal biopsy registry, initiation of national and state level screening programme for renal diseases and early identification of urinary abnormalities and prompt referral to nephrologist for renal biopsy.

LIMITATIONS OF THE STUDY

As this study is done in small group of patients in a single centre, a multicentre large study is needed for more clarification and better results. As extreme age group and post-transplant patients are excluded from this study, these results are not applicable for these categories. As I have mentioned earlier various studies in various part of the world reported the prevalence of the primary and secondary glomerular diseases in their institutes or states. So it is very difficult to delineate the changing pattern of the renal diseases. Because of increasing biopsy rate, changing trend of the renal diseases and ethnical racial variability, the prevalence of various diagnosis in renal biopsy results are highly evolving over the time.

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ANEXURES

NAME OF THE PATIENT:	OCCUPATION
AGE:	SMOKER/NON-
SMOKER	
SEX:	ALCOHOLIC/NON-
ALCOHOLIC	
IP NO:	
DATE OF ADMISSION:	
DATE OF DISCHARGE:	

EDEMA
OLIGURIA
HEMATURIA
DYSPNOEA

PRIOR RENAL DISEASE
HT-DURATION/COMPLICATION
DM-DURATION/COMPLICATION
DYSLIPIDEMIA
CAD
HYPOTHYROIDISM
SLE
AMYLOIDOSIS

NSAIDS
AMINOGLYCOSIDES
STEROIDS
OHA
INSULIN
AHT
HIV STATUS
VIRAL MARKERS STATUS
HBV
HCV
ANA
ANTI DsDNA
COMPLIMENTS

EXAMINATION FINDINGS

PALLOR

SKIN LESIONS

ANASARCA

BP

INVESTIGATIONS

URINE-PROTEIN

SUGAR

DEPOSITS

CBC

BLOOD SUGAR

UREA

CREATININE

SERUM ELECTROLYTES

LIPIDS

VIRAL MARKERS

OTHERS

USG-KUB

ECG

ECHO

CLINICAL DIAGNOSIS

THERAPY

DIALYSIS

RECOVERY

DEATH

BIOPSY REPORT

FINAL DIAGNOSIS

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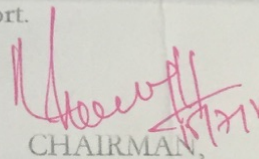
Protocol ID. No.04/04/2015 Meeting held on 09/04/2015

CERTIFICATE OF APPROVAL

The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for approval "Clinical and histopathological profile in patients undergoing renal biopsy in a tertiary care centre"- For Dissertation Purpose" submitted by Dr.S.Settu, Post Graduate in MD (GM), Govt. Kilpauk Medical College, Chennai.

The Proposal is APPROVED.

The Institutional Ethical Committee expects to be informed about the progress of the study any Adverse Drug Reaction Occurring in the Course of the study any change in the protocol and patient information /informed consent and asks to be provided a copy of the final report.

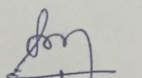


CHAIRMAN,

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CLINICAL AND HISTOPATHOLOGICAL PROFILE IN PATIENTS UNDERWENT RENAL BIOPSY IN A TERTIARY CARE CENTRE

Dissertation submitted to

THE TAMILNADU DR. M.G.R MEDICAL UNIVERSITY
CHENNAI

In partial fulfillment of regulations

For award of the degree of

M.D (GENERAL MEDICINE)

BRANCH - I



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